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VOLUME 21 (old series Volume 78) JANUARY 1939 NUMBER 1

CORRECTION OF PTOSIS BY ATTACHMENT OF STRIPS OF ORBICULARIS MUSCLE TO THE SUPERIOR RECTUS MUSCLE

JOHN M. WHEELER, M.D.†
NEW YORK

In the surgical treatment of ptosis corrective effects are dependent on three different principles: (*a*) dependence on the attachment of the frontalis muscle to the lid by cicatricial bands, strips of skin, strips of orbicularis muscle and fascia lata; (*b*) shortening of the levator muscle of the upper lid by means of resection of the tarsus and resection or tucking of the levator muscle, or (*c*) dependence on elevation of the globe by means of the Motais operation and its modifications, the Young [1] operation, in which the upper lid is made to adhere to the globe, the Dickey [2] fascia lata sling or the Trainor [3] procedure, in which a strip of tarsus is passed under the superior rectus muscle.

Operations that depend for effect on the attachment of the lid to the frontalis muscle are not to be recommended for two chief reasons: First, the direction of pull is forward and upward instead of backward and upward, as is the case normally; and second, the elevation of the lid is accompanied by elevation of the brow and wrinkling of the forehead, with the effect of effort and strain.

Shortening of the levator muscle is the operation of choice if the ptosis is slight and if the lid has ability to move with the globe, even though the palpebral fissure may be too narrow. Resection of the levator muscle increases the effectiveness of its action.

The operations that cause the lid to follow the globe are necessary for drooping of the lid of considerable amount, if the lid does not tend to follow the elevation of the globe. In such a case the palpebral fissure may be actually narrower when the patient looks up than when he

† Dr. Wheeler died Aug. 22, 1938.

Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 15, 1938.

1. Young, G.: Operation for Congenital Ptosis, Brit. J. Ophth. **8**:272 (June) 1924.

2. Dickey, C. A.: Superior-Rectus Fascia-Lata Sling in the Correction of Ptosis, Am. J. Ophth. **19**:660 (Aug.) 1936.

3. Trainor, M. E.: Operation for Lid Ptosis, Tr. Sect. Ophth., A. M. A., 1935, p. 93.

looks down, for the margin of the lower lid may follow after the globe in its upward movement while the upper lid remains stationary.

The Motais operation, especially with modifications introduced by Shoemaker [4] and Kirby,[5] gives striking results, but it has four objections: First, there is a tendency toward angulation of the margin of the upper lid from the sharply localized attachment of the little strip of superior rectus muscle. Second, the superior rectus muscle is disturbed and injured. Third, the dissection entailed is considered difficult. Fourth, rarely, near the attachment of the superior rectus muscle to the upper lid entropion may occur and cilia rub on the cornea.

DESCRIPTION OF OPERATION

Anesthesia.—Avertin with the injection of procaine hydrochloride works well. The subcutaneous infiltration near the margin of the lid helps in the dissection because it magnifies the rather thin layer of the orbicularis muscle overlying the tarsus.

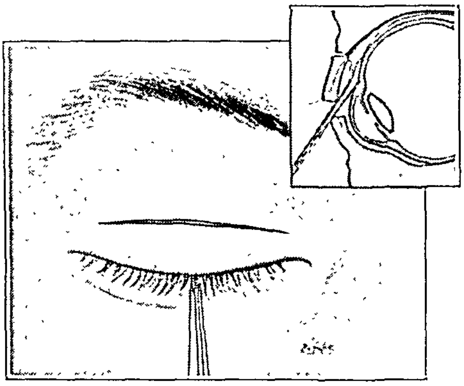

Fig. 1.—Primary incision. The inset attempts to show how the globe and the conjunctiva of the fornix are pulled down to facilitate the dissection.

The incision in the skin is made at the level of the upper border of the tarsus, and is about 25 mm. long (fig. 1).

The skin is dissected up from the orbicularis muscle nearly to the margin of the lid. (Care should be taken not to injure the cilia follicles.) The subcutaneous dissection is then carried upward about 10 mm.

A horizontal incision is made through the orbicularis muscle 4 or 5 mm. above the tarsus and carried through the tarso-orbital fascia and the levator tendon. Then the dissection is carried through Tenon's capsule to the sclera on each side of the tendon of the superior rectus muscle. This muscle is picked up on a squint hook, and its superior surface is exposed. During this dissection the globe is held in depression by a suture which has been passed through the conjunctiva and the superficial sclera just above the cornea. Another suture is carried through the conjunctiva of the upper fornix, and traction drags the upper cul-de-sac down out of the way of the dissection (fig. 1 inset and fig. 5).

4. Shoemaker, W. T.: Observations of the Motais Operation for Ptosis: Report of Three Cases, Ann. Ophth. **16**:608, 1907.

5. Kirby, D. B.: Modified Motais Operation for Blepharoptosis, Arch. Ophth. **57**:327 (July) 1928.

Strips of orbicularis muscle are dissected up from the surface of the tarsus. They are set free at the ends toward the canthi but are left attached at the ends toward the center of the lid. The attached ends are about 8 mm. apart, and the orbicularis muscle between is undisturbed. Each strip is about 10 mm. long and 4 mm. wide (fig. 3).

The strips of orbicularis muscle are attached to the upper surface of the superior rectus muscle with 000 chromic catgut. It is well for each strip to be attached by two sutures, as the strips have to carry a heavy load during the healing process when the upper lid is swollen.

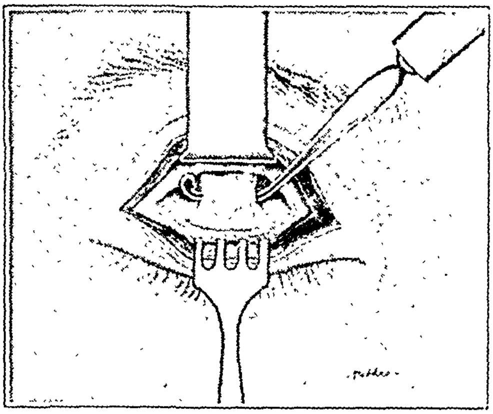

Fig. 2.—The dissection above the tarsus has been carried through the orbicularis muscle, the tarso-orbital fascia, the levator muscle and Tenon's capsule. The superior rectus muscle is held on a hook.

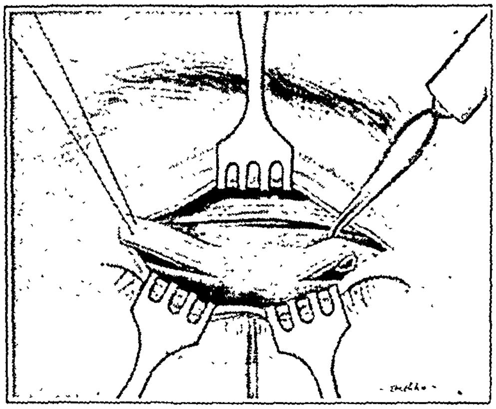

Fig. 3.—Strips of orbicularis muscle 4 mm. wide and 10 mm. long are dissected up from the tarsus, and the ends toward the canthi are cut free. The attached ends are about 8 mm. apart.

An ordinary probe passed between the superior rectus muscle and the sclera and allowed to project out of the dissected area on each side is useful in steadying the globe in depression while the strips of orbicularis muscle are being attached.

After the orbicularis muscle is secured in contact with the superior rectus muscle, the only suturing required is that of the skin. This can be closed by several fine silk sutures or by a single subcutaneous suture.

Figure 4 shows the attachment of the strips of orbicularis muscle to the surface of the superior rectus muscle, and figure 5 shows the attachment in cross section.

Dressing.—Injury to the partially exposed cornea after operation must be avoided. The dressing should be such that the cornea is properly protected. For several years I have used a special dressing on account of lagophthalmos after the Motais type of operation, and this dressing can be recommended. A thin layer of absorbent cotton is dipped in warm water and then pressed almost dry between the palms of the hands. This is shaped to make a small cone, with the base large enough to cover the base of the orbit. The cone is filled with sterile petrolatum and placed over the eye and lids. Then another layer of moist

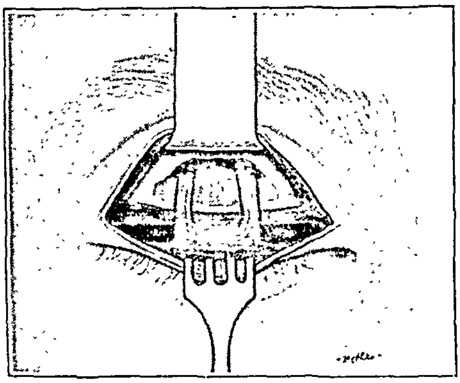

Fig. 4.—The strips of orbicularis muscle are attached to the superior rectus muscle with fine catgut.

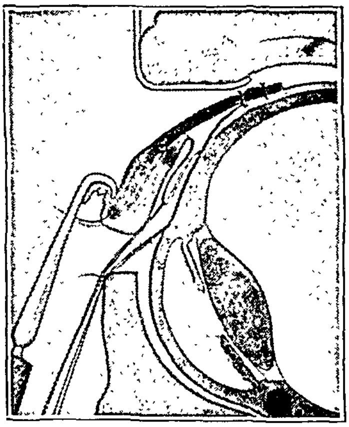

Fig. 5.—Cross section to show attachment of the orbicularis muscle to the superior rectus muscle. The globe and the upper conjunctival cul-de-sac are held down by sutures.

cotton is placed over the cone with the overlapping joint in a position not corresponding with that of the first layer of the cone. Still a third thin layer is put over the cone. Strips of adhesive plaster are built up from the base to the apex of the cone, to hold it in place and to strengthen it. Even if a child should lie on such a dressing, it is secure enough protection so that nothing but petrolatum could touch the eye. This should be left on for about a week. After it is removed, petrolatum should be put in the palpebral fissure before the patient goes to sleep, as long as lagophthalmos persists.

COMMENT

In my experience with the operation there has been a tendency toward undercorrection, as there has been in other operations for ptosis. Probably a good rule is for the surgeon to leave the margin of the upper lid 2 or 3 mm. above the limbus at the close of the operation. In no case has there been overcorrection of the ptosis.

Moderate temporary lagophthalmos follows the operation and gradually disappears over a period of a few weeks.

A normal uninterrupted fold of skin develops in good position, with a covered crease at the line of incision along the upper border of the tarsus.

There is no tendency toward angulation of the margin of the lid from the pull of the two strips of orbicularis muscle, nor is any other deformity of the lid caused by the operation.

Impairment of elevation of the eyeball follows the operation, but as the postoperative reaction subsides the upward movement of the eyeball improves almost to normal.

Considerable swelling of the upper lid follows the operation, so that the eye may be almost closed for several days, but in no case has the attachment of the strips of orbicularis muscle to the superior rectus muscle been destroyed. As the swelling disappears, the movement of the upper lid develops.

The contour of the upper lid remains good, without a suggestion of angulation.

An objection to the operation is that the dissection might be considered difficult.

635 West One Hundred and Sixty-Fifth Street.

ABSTRACT OF DISCUSSION

Dr. John O. McReynolds, Dallas, Texas: I have performed the operation as described by Dr. Wheeler with some modifications. I approach the tendon more directly in its place of attachment on the upper surface of the globe. After separating it freely from the bulb, I carry a strong silk thread under the tendon and use this bridle rein or halter rein as a substitute for the two threads that Dr. Wheeler introduced into the superior retrotarsal fold and into the episcleral tissue above the cornea. Then, instead of reaching down for the tendon through the cutaneous and fascial incision, I grasp the halter rein with a forceps and carry it upward through the incision until the superior rectus muscle presents in plain view through and above the incision.

Dr. Wheeler's operation does not involve section of any of the fibers of the superior rectus muscle and leaves the tendon in its entirety still attached to the globe with its power unimpaired. After the first postoperative day, the controlling suture in the upper lid can be

eliminated, and upward traction on the suture in the lower lid will be sufficient to keep the cornea covered and protected adequately by the updrawn lower lid.

Dr. John Weeks, Portland, Ore.: On a recent visit to New York I had the privilege of watching Dr. Wheeler perform his operation. Ptosis is usually congenital. It may be acquired and may or may not be associated with paresis or paralysis of the extraocular muscles. The operation desirable in any case depends on the conditions attending the individual patient. If the superior rectus muscle does not function, one must depend on attachment to the lower anterior border of the occipitofrontalis muscle to obtain movements of the upper lid. This may be done either by skin flaps, of which the Panas operation is an example, or by the use of portions of the orbicularis palpebrarum muscle, as was employed by the late Dr. Robert Reece. The portions of the orbicularis muscle raised by Dr. Reece were similar to those that were raised by Dr. Wheeler; the portions were carried under the skin to the anterior margin of the occipitofrontalis muscle and there attached. If pronounced movement of the lid is not necessary and the ptosis is not great, resection of the tendon of the levator muscle may suffice, or a shortening of the upper lid may be done after the manner of Gillet de Grandmont, by which procedure a widening of the palpebral fissure from 2 to 4 mm. may be obtained without endangering the closure of the eyes during sleep.

If the superior rectus muscle functions normally, the principle of attachment of the lid to this muscle may be satisfactorily applied, combined in cases in which the tissues of the lid are exceptionally heavy with removal of some of the tissue of the lid, or an undercorrection may be corrected by the operation of de Grandmont. The Motais operation has given good results in the cases in which I have applied it. The modification of the Motais operation by Shoemaker and by Kirby are valuable aids in the technic.

In the operation described by Dr. Wheeler the strips of orbicularis muscle were raised much as was done by Dr. Reece, but instead of being attached to the occipitofrontalis muscle, they were attached to the superior rectus muscle. The difficulties in technic are apparently about the same as in the Motais operation. If the power of the superior rectus muscle is better retained by the operation described by Dr. Wheeler than by the Motais operation, Dr. Wheeler's operation should be the one of choice.

Dr. C. A. Dickey, San Francisco: As the time is limited, I should like to show some slides of an operation I devised about four years ago. While I dissect out the superior rectus muscle, an assistant obtains a strip of fascia lata. The superior rectus muscle is isolated, and the central half of the muscle is separated. The fascial strip is inserted under the central half, and the capsule and conjunctiva are sutured with quadruple 0 catgut.

The incision is made in the lid as Dr. Wheeler does, and the tarsal plate is exposed fairly well down toward the margin of the lid. An incision is made at the upper border of the tarsus, and the strip of fascia lata is drawn through. Two white silk sutures are placed in the tarsus, one at the junction of the outer and the middle third of the

tarsus and the other at the junction of the inner and the middle third. One suture is passed through the fascia and tied. The length of the fascial sling required to produce the proper elevation of the lid is then estimated or, in other words, the length of the sling determines the position of the lid. When this is decided, the fascia is tied down to the tarsus, and the incision in the skin is closed with a subcuticular stitch.

The advantages of this procedure are that any degree of elevation may be obtained, and in the event of a failure the operation may be repeated easily, as there has been no damage done either to the superior rectus muscle or to the lid. Also, the full action of the superior rectus muscle is utilized, as Dr. Jackson pointed out when I first presented this procedure three years ago.

Dr. W. D. Horner, San Francisco: Dr. Wheeler and Dr. Dickey apparently have presented a good substitute for the Motais operation, which, while an old favorite, has certain defects, which Dr. Wheeler pointed out. The new operations correct these difficulties by a substitution of a two point suspension of the lid instead of the one offered by the Motais. There is no permanent damage to the superior rectus muscle by either. This means a good deal, because not every operation for ptosis gives satisfactory results, and sometimes it is necessary or worth while to operate a second time. This can be done, I think, with either method.

My interest in Dr. Dickey's operation has been keen, naturally, because it was developed at the ophthalmic clinic of the University of California Medical School, and I have had the opportunity of seeing him work and also have had the opportunity of doing the operation myself. I have performed the operation in about 20 cases altogether, and the results have been satisfactory. One, however, must remember that all textbooks end up or begin the discussion of ptosis with the words that operative treatment of ptosis is inherently difficult.

Dr. John M. Wheeler, New York: I am pleased that Dr. McReynolds considered my operation important enough to modify it. I think that there may be certain advantages in keeping out of the conjunctival sac, so that personally I should prefer to operate through the sort of incision that I have made.

Dr. Dickey's operation is interesting, but I have not performed it. I should think that the results would be much the same with the two procedures, but in doing his operation I think that an unnecessary dissection is made in the thigh. If suitable material can be found in the lid, it seems as though it would be preferable for the surgeon to use such material. Dr. Dickey in an article in which he described his operation spoke of the fascia lata giving way in 1 case, and I think that in that case the fascia had been preserved. Evidently now he believes that a dissection in the thigh should be made in order to obtain the tissue necessary for suspension.

I should like to repeat that in case the eyelid makes a worthy attempt to go up with the eyeball, I should not recommend this operation but suggest increasing the effectiveness of the levator muscle by shortening it. This operation, I think, should be done only in case the lid fails entirely to go up with the eyeball.

ROLE OF NONVIOLENCE IN LEVER ACTION INTRACAPSULAR EXTRACTION OF CATARACT

KARTIC CHUNDER DUTT
Chief Medical Officer of the Sonpur State
SONPUR RAJ, INDIA

Since the publication of my article on the lever action operation for the intracapsular extraction of cataract in the ARCHIVES [1] letters of inquiry about further particulars of this method and the special instruments employed, viz., the hyalonavicular fulcrum and the mango leaf dislocator, have been received. In the previous article the scientific principle of lever action and its technical application to the extraction of cataract intracapsularly were described. It now remains to explain what makes this method safer, surer and at the same time simpler than capsulotomy and the other intracapsular methods. The secret of success of the intracapsular extraction of cataract by the lever action technic lies in the adaptation of the principle of nonviolence in its performance—thanks to Mahatma Gandhi, whose principle of nonviolence is applicable to cataract extractions even more appropriately than to political movements. The reason for the success of nonviolence in cataract extraction has been well expressed by J. W. Wright: [2] "There is no organ of the entire body so sensitively adjusted comparable to the eye, being composed of various intricate structures liberally supplied by one-half of the cranial nerves" (from the second to the seventh cranial nerve) and, moreover, "each structure is endowed with functional activities" of a delicate nature which are impaired by the slightest trauma, ectopia or irritation or inflammation of the sensitive structures. Who can doubt that an operation for the extraction of cataract on such a delicate and sensitive organ should be strictly nonviolent in order to insure that no trauma or irritation is done at the time of operation and that no inflammatory reaction follows? Nowadays, irritation, trauma, ectopia and mutilation are more or less enacted under cover of local anesthesia, facial akinesia and retrobulbar anesthesia in all the ingenious and complicated modern methods of cataract extraction. Though the anesthetized eye seems to feel the same at the time of operation, the postoperative results prove that these irritations are not to be tolerated with impunity by this most delicate and sensitive organ of the body. Hence, what

1. Dutt, K. C.: Lever Action Operation for Intracapsular Extraction of Cataract, Arch. Ophth. **18**:897 (Dec.) 1937.
2. Wright, J. W.: Columbus M. J. **3**:145, 1884.

is often found in such cases is the striking paradox of a most successful extraction of cataract by ingenious instrumentation and ultimate results which are not successful. This fact is too significant to be overlooked. What is the lesson it carries? Colonel Smith's [3] experience and observation point to the reason. Smith [3c] wrote: "As a matter of fact the outer surface of the cornea will tolerate any amount of massaging without showing any reaction. It is the under surface which is intolerant of instrumentation. Hence other things being equal, the less we introduce instruments into the eye the better." To insure successful results and an uneventful postoperative period, lever action intracapsular technic, among other things, observes strict nonviolence to the eye and especially to its internal structures. But regardless of how delicate the operative technic is, there is always the problem of trauma to the tissue. Lever action has solved this difficult problem so far as is possible and has worked out the irreducible minimum of violence required for the extraction of cataract. In ordinary cases this technic consists of (1) a limbic incision into the upper half of the sclerocorneal arc and (2) delivery of the lens in its capsule by lever action with a minimum of force or effort, applied from the outer surface of the eye. In such cases iridectomy has been eliminated and prolapse of the iris prevented by postural treatment. The scientific principle of lever action has been applied so as to insure a mechanical advantage by which much less effort is required to dislocate and rotate the lens in capsule and still less is required for the detachment of the zonule than the actual strength of anchorage of the zonule. As this minimum of effort is applied from the outer surface of the eye and in a direction forward and upward away from the vitreous and other internal structures of the eye, the objective of an irreducible minimum of violence has been attained. Consequently, lever action intracapsular extraction is universally applicable to all the varieties of cataract in all the stages of formation and with varying degrees of strength of attachment of their zonules without risk of loss of vitreous or rupture of the capsule.

W. A. Fisher [4] in his article on senile cataract stated: "The intracapsular operation for senile cataract is becoming daily more popular and operators are looking for a method whereby vitreous loss and ruptured capsules together with other complications due to these accidents will be reduced to a minimum." Indeed, the time is fast approaching when neither the patient nor the surgeon can afford to wait for

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3. Smith, H.: (*a*) Arch. Ophth. **33**:64, 1904; (*b*) **55**:213, 1926; (*c*) Tr. Ophth. Soc. U. Kingdom **45**:123, 1925; (*d*) Ophth. Rec. **19**:54, 1910; (*e*) Treatment of Cataract and Some Other Common Ocular Affections, ed. 2, London, Butterworth & Company, 1928, p. 167.

4. Fisher, W. A.: Senile Cataract: Methods of Operating, ed. 3, Chicago, H. G. Adair Printing Co., 1937, p. 134.

the indefinite periods necessary for the ripening of different varieties of cataract. Consequently, as soon as, or soon after, useful vision is lost the cataract should be extracted. Therefore, either the cataract is extracted intracapsularly or capsulotomy is performed before the cataract is fully mature. But capsulotomy performed during immaturity of the cataract renders the expression of the cortical matter of the lens difficult, even after repeated irrigation of the anterior chamber. The new additions to the technic of capsulotomy, viz., many bizarre incisions in the capsule; preliminary capsulotomy; opening the capsule with a sharp-toothed forceps or with a blunt-toothed forceps; conjunctival flaps; conjunctival sutures; corneal, corneoconjunctival, corneoscleral and conjunctivoscleral sutures with facial akinesia and retrobulbar anesthesia; bridal sutures, and irrigation of the anterior chamber by means of various types of irrigators and different lotions, have not yielded better results than the original technic of Daviel. Early extraction in the premature stage, combined with the aforementioned complicated and prolonged operations, acts rather adversely, by the formation of iritis and an after-cataract which is too dense to be dealt with by needling. Hence, the needling has to be repeated. But repeated needling is likely to give rise to endophthalmitis phacoanaphylactica due to the distintegration of lens protein which acts as foreign matter and as a toxin. Thus, Fisher [5] wrote: "Needling operations are considered by many as being quite dangerous, always leaving a hernia of the vitreous, and are often followed by post-operative inflammations and too often by inflammation with great danger of serious loss of vision." Smith [6] in his book on the treatment of cataract stated: "For the surgeon who extracts cataract by capsulotomy is now-a-days forced by the pressure of competition to extend his sphere of action to the immature stage. The density and toughness of the after-cataract which ensues drive him to needle it while it is still young and tender, before the eye has lost its intolerance to further operative interference and has recovered a stable equilibrium." Hence, "sudden disasters which frequently destroy the eye" follow.

In this connection, Duke-Elder [7] stated: "It is well known that after an extracapsular extraction of cataract, especially when a considerable quantity of lens material is left in the eye, an iritis, sometimes of considerable severity, is commonplace. The theory that the reaction in the eye is due to an anaphylactic reaction to the lens proteins is already well known from the work of Burky and Woods,[8] as is also

5. Fisher,[4] p. 136.

6. Smith,[3e] p. 188.

7. Duke-Elder, S., in Tidy, H. L., and Short, A. R.: Medical Annual, Bristol, England, John Wright & Sons, Ltd., 1936, p. 112.

8. Burky, E. L., and Woods, A. C.: Lens Extract: Its Preparation and Clinical Use, Arch. Ophth. 6:548 (Oct.) 1931.

their suggestion that sensitive patients should be desensitized before an operation is undertaken." Efforts have been made by Goodman[9] to minimize anaphylactic reaction by previous injections of graduated doses of lens protein.

In spite of the aforementioned recent advances toward the improvement of capsulotomy and its after-effects, i. e., postoperative inflammation of the iris and ciliary body, blurred vision, preliminary injections of lens protein, formation of after-cataracts, risks of repeated needling, sensitization and irritability of the eye, all these drawbacks still remain as stumbling blocks to the universal application of this method for the extraction of cataract. In addition, there are hard cataracts with such thick capsules that they cannot be properly cut with the cystitome. These short-comings attend capsulotomy regardless of all attempts to remedy them. The newer additions to the technic have rather added to the complications and made matters worse than before.

On the other hand, many ophthalmic surgeons in India, America and Europe have acquired skill and experience with the different methods of intracapsular operation and have established their name and fame with the operation of their choice. This is especially practicable now because the old prejudices and apprehensions against any of the intracapsular methods no longer exist. Each intracapsular method is put to practice by every up-to-date ophthalmic surgeon in suitable cases.

Formerly, some advocates of extraction by capsulotomy contended that the after-effects of intracapsular extraction were not good. In order to find out the truth of this assertion, some eminent surgeons took pains to investigate carefully the end results in cases in which intracapsular extraction was used.

The investigations and observations were carried on by Lister,[10] Knapp,[11] Fuchs, Fisher,[12] Pratt,[13] Greene,[14] Greene and Millette,[15]

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9. Goodman, E. L.: Endophthalmitis Phaco-Anaphylactica, Arch. Ophth. **14**: 90 (July) 1935.

10. Lister, A. E. J., in Smith,[3a] pp. 242-258.

11. Knapp, A.: Arch. Ophth. **44**:1, 1915; Tr. Ophth. Soc. U. Kingdom **45**: 117, 1925.

12. Fisher, W. A.: Arch. Ophth. **44**:18, 1915; J. Ophth. & Oto-Laryng. **4**:379, 1910; Ophthalmology **11**:318, 1915; Ann. Ophth. **24**:718, 1915; **26**:79, 1917.

13. Pratt, F. J., and Pratt, J. A.: Minnesota Med. **4**:370, 1921.

14. Greene, D. W.: Intracapsular Extraction of Cataract, in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1914, vol. 3, p. 1533; Experience in the Expression of Cataracts in the Capsule by the Smith Method, Tr. Sect. Ophth., A. M. A., 1909, p. 177.

15. Greene, D. W., and Millette, J. W.: Visual Results After the Smith Operation for Cataract, J. A. M. A. **59**:1070 (Sept. 21) 1912.

Elschnig [16] and others in cases in which Smith's pressure method and the Knapp-Török [17]-Elschnig method, which employed traction with a forceps combined with pressure, were used. The results of their investigations have definitely proved that removal of the posterior capsule does not produce degenerative changes in the vitreous, or glaucoma or detachment of the retina.

Again, a systematic follow-up study and a minute investigation of the end results were carried out by Vail,[18] A. S. and L. D. Green,[19] Urra,[20] Cruickshank,[21] Cadilhac,[22] Saint-Martin,[23] Wright [24] and others in cases in which intracapsular extraction was done by Barraquer's vacuum suction method and by vacuum suction combined with pressure. The results of these investigations have established the facts that there are few complications and that the vitreous gel does not show opacities or any other pathologic change afterward.

These truth-seekers have done a great service to suffering humanity by removing the apprehensions of opacification or degeneration of the vitreous at a later date after intracapsular extraction of cataract and by proving that the after-complications are fewer than after extraction by capsulotomy.

It has therefore been possible for the surgeon to operate by any of the intracapsular methods as may be deemed suitable in a particular case. The patient has also been relieved of the anxiety about the after-complications and can now implicitly rely on the discretion and judgment of the surgeon as to the selection of the method of operation.

Although the prejudices and the apprehensions against intracapsular extraction have been removed, why is it that this method cannot be universally applicable? Duke-Elder [25] cleared up this point in his textbook on the recent advances in ophthalmology: "In these days, newer and more complicated procedures are being devised and advocated for the operative technique of the extraction of cataract, all of them intro-

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16. Elschnig, A.: Die intrakapsulare Starextrakation, ed. 2 and 3, Berlin, Julius Springer, 1932; Ztschr. f. Augenh. **75**:1, 1931; Am. J. Ophth. **8**:355, 1925.

17. Török, E.: Ann. Ophth. **25**:712, 1916.

18. Vail, D. T.: Ophth. Rec. **24**:248, 1915; Arch. Ophth. **45**:307, 1916.

19. Green, A. S., and Green, L. D.: Intracapsular Extraction of Senile Cataract, Tr. Sect. Ophth., A. M. A., 1917, p. 313; Am. J. Ophth. **5**:92, 1922; Arch. Ophth. **51**:338, 1922.

20. Urra, M.: España oftal. **6**:101, 1921.

21. Cruickshank, M. M.: Brit. J. Ophth. **9**:321, 1925.

22. Cadilhac, G.: L'extraction totale de la cataracte par l'erisiphaque, Paris, Masson & Cie, 1930.

23. Saint-Martin, cited by Cadilhac.[22]

24. Wright, R. E.: Am. J. Ophth. **7**:155, 1924.

25. Duke-Elder, S.: Recent Advances in Ophthalmology, ed. 3, Philadelphia, P. Blakiston's Son & Co., 1934.

ducing more manipulative difficulties at the time of operation and most of them more dangerous afterwards." No doubt Duke-Elder had the right idea. Indeed, the tendency toward devising complicated procedures and inventing complicated instruments for the performance of cataract extraction has proved to be unprofitable in the long run, so far as the end results of the operation are concerned. More and more ingenious and complicated instruments are finding a place in the list of new inventions. Barraquer's [26] erisphake, invented in 1917, is really an ingenious and complicated instrument with complicated machinery which is difficult to work at a constant vacuum negative pressure during the whole period of the operation. Sometimes, the erisphake gets out of order in the middle of the operation. Moreover, when it is out of order the instrument cannot be readjusted immediately, because it is too hot to handle at that time. Lacarrère's [27] diathermic coagulation needle, invented in 1932, is even more ingenious and more difficult to control. It requires much skill to insure that only the necessary degree of coagulation of the lens protein is effected and that coagulation of the underlying vitreous is not accidentally done. The technics of these operations as well as those of the other combined and complicated methods look well on paper and also display the skill and dexterity of the surgeon at the time of operation, but when the time for reckoning comes the results belie all sanguine expectations. The end results are far from satisfactory! All the labor is lost!

Is it not time to call a halt to the further invention of complicated technics and ingenious instruments? Would it not be better to invent simpler instruments which can never get out of order, which will work from the outside and which will inflict no violence and will not irritate the delicate internal structures of the eye? Would it not be better to devise a method which is technically easy for the surgeon, scientifically advantageous and profitable and practically easy for the eye to bear? Is it not better to devise a technic which is universally applicable to all forms of cataract in all the stages of formation without fail—a technic which is free from the risk of the surgeon having to revert to capsulotomy in the middle of the operation; which is free from the risk of loss of vitreous and rupture of the capsule at the time of its performance, and which is free from risk of postoperative inflammation? All the aforementioned conditions have been satisfied by the lever action intracapsular technic.

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26. Barraquer, I.: (*a*) Brit. M. J. **2**:660, 1924; (*b*) Arch. de oftal. hispano-am. **17**:252, 1917; (*c*) Arch. Ophth. **50**:307, 1921; (*d*) **51**:448, 1922; (*e*) Facoerisis, in Fisher,[4] p. 8.

27. Lacarrère, J. L.: Klin. Monatsbl. f. Augenh. **88**:778, 1932; Arch. de oftal. hispano-am. **32**:293, 1932.

The scientific basis of the lever action intracapsular operation renders the technic safer and surer than the application of the force by expression, as with capsulotomy, or by pressure, by suction, by traction or a combination of these forces, as with the intracapsular methods. This is because lever action acts at a mechanical advantage which is always greater than the original force employed and therefore is always mechanically more advantageous and profitable than any of the other methods. It has been proved by mathematical equations in my previous article[1] on this operation that the resulting available power for the dislocation and rotation of the lens-in-capsule and for the detachment of the zonule is always greater than the power originally employed by the surgeon's hand. This advantage of "profit of power" which characterizes the lever action intracapsular method is not available with capsulotomy, in which the power employed consists of expression, or with any of the intracapsular methods, in which the power employed consists of pressure, suction, traction, suction with pressure or traction with pressure. This makes lever action safer and surer and at the same time easier and more profitable than all other methods of cataract extraction, capsulotomy or intracapsular extraction.

Moreover, lever action is performed by instruments which are simple in their construction, really imitations of nature in their formation, are easy to manipulate and never get out of order. The mango leaf dislocator, the hyalonavicular fulcrum and the stirrup eyelid retractors will find favor with any surgeon who pleases to practice with them.[28] Photographs of these instruments appeared in my previous article.[29]

It is to be especially noted that the hyalonavicular fulcrum is a double-headed silver instrument. In fact, two fulcrum instruments, viz., the hyaloid socket fulcrum and the navicular socket fulcrum, have been combined in the double head, which is the part of the instrument that does the work. Both the socket fulcrums have been made in imitation of nature. The hyaloid socket has been made in imitation of the hyaloid fossa in the vitreous, and the navicular socket, in imitation of a 5 mm. section of the curve of the socket within the ciliary body into which the equator of the lens-in-capsule is socketed by nature. The reason for this combination is as follows: If two separate instruments, the hyaloid socket fulcrum and the navicular socket fulcrum, were made, the operator would be considerably inconvenienced, because the 12 o'clock upper convex border of the equator of the lens-in-capsule is socketed into the concavity of the navicular socket fulcrum during the first and second stages of the operation, viz., stage I, consisting of dis-

28. These instruments may be obtained from Down Bros., Ltd., London.

29. Dutt,[1] p. 900, fig. 2.

location of the lens-in-capsule, and stage II, consisting of rotation of the lens-in-capsule. The reader is referred to my previous article for a diagram illustrating stage I.[30]

The concavity of the navicular socket fulcrum is kept applied to this portion of the lens-in-capsule during stage II of the operation. A diagram illustrating this stage was also reproduced in my previous article.[31]

During stage III, that of detachment of the zonule, the convexity of the anterior surface of the lens-in-capsule is socketed within the concavity of the hyaloid fulcrum. If two instruments were used, the second instrument, the hyaloid socket fulcrum, would have to be taken from the instrument tray. This change of instrument in the middle of the operation would necessitate postponement of the operation for the time being. To obviate this postponement when the operation is half done, the combined double-headed instrument was devised and is used with convenience. A diagram illustrating stage III is to be found in my previous article.[32]

As soon as the second stage of rotation of the lens-in-capsule is over, the surgeon has only to turn the combined instrument, i. e., the hyalonavicular fulcrum, between his thumb on one side and the other fingers on the other side in order to change the navicular socket fulcrum and to engage the hyaloid socket fulcrum. This simple maneuver at once engages the anterior pole and the anterior surface of the lens-in-

30. Dutt,[1] p. 901, fig. 3. In this diagram are shown the navicular fulcrum, applied at 12 o'clock outside the sclera; effort or power applied with the tip of the mango leaf dislocator at 6 o'clock, outside the sclera, and points of resistance (*R* and *R'*) of the zonule to be overcome, inside on each side of 6 o'clock. By this procedure the more or less rigid cataractous lens-in-capsule is converted into a lever. In this lever the resistance of the zonule is placed between the fulcrum and the effort; hence, a lever of class II is formed. A diagram illustrating this lever appears on page 898 of the article just mentioned.

31. Dutt, p. 902, fig. 4. In this illustration are shown the navicular fulcrum applied at 12 o'clock, socketing the upper border of the lens-in-capsule; effort applied steadily forward by insinuating the tip of the mango leaf dislocator at 6 o'clock on the breach of the zonule from the outside to hitch the lower posterior border of the equator of the lens-in-capsule so as to tilt it forward, and the points of resistance (*RR'*) in the attachment of the zonule. When *RR'* give way, the resistance recedes to points *R''* *R'''* in the zonular attachment. The lower border of the lens-in-capsule is rotated gradually forward and upward as more and more of the zonule is peeled off.

32. Dutt,[1] p. 903, fig. 5. This illustration shows the hyaloid socket fulcrum applied at 12 o'clock and the bend of the mango leaf dislocator applied behind and below the lower border of the lens-in-capsule. The remaining portion of the zonule from 6 to 12 o'clock from the points of resistance *R''* *R'''* up to 12 o'clock is detached gradually as the cataractous lens-in-capsule turns on the hyaloid socket fulcrum.

capsule within the hyaloid socket of the hyalonavicular fulcrum. The anterior convex surface of the lens-in-capsule automatically fits into the hyaloid socket nicely and cannot slip out. The lens is hereafter conveniently held between the hyaloid socket fulcrum on the anterior side and the bend of the mango leaf dislocator on its posterior side.

As has been stated previously, the navicular socket fulcrum has been made in imitation of a 5 mm. section of the curve within the ciliary body into which the equator of the lens-in-capsule is socketed by nature. The navicular socket is carved longitudinally on one face of the head of the hyalonavicular fulcrum. The socket is a boat-shaped furrow about 0.5 mm. in depth in the central part, while the two sides gradually diverge and form the crest. Thus, a shallow concavity is formed. When the navicular socket fulcrum is placed horizontally and tangentially to the optic globe at 12 o'clock on its surface, the convex upper border of the lens-in-capsule at its equator is socketed within the navicular socket fulcrum, padded by the sclera. The convex upper border of the equator of the lens-in-capsule may thus freely turn within the navicular socket fulcrum in a hingelike fashion and cannot slip out.

The hyaloid socket, which is made in imitation of the shape and size of the hyaloid fossa of the vitreous, consequently presents a uniformly round and hyaloid-fossa-like concavity about 0.5 mm. in depth and 5 mm. in diameter. It may be here noted that the hyaloid fossa of the vitreous accommodates the posterior convex surface of the lens-in-capsule, whereas the hyaloid socket fulcrum is intended to accommodate the anterior convex surface. But it is an anatomic fact that the anterior surface of the lens-in-capsule is a bit less convex than the posterior surface. Accordingly, the hyaloid-fossa-like concavity of the hyalonavicular fulcrum is a bit less concave than the hyaloid fossa of the vitreous. When in action during the third stage of detachment of the zonule, the hyaloid socket fulcrum accommodates the anterior convex surface of the lens-in-capsule so as to form something like a ball and socket joint fulcrum into which the anterior convex surface of the lens can freely turn by lever action.

The mango leaf dislocator is a blut-pointed, lanceolate, leaf-shaped instrument, 4 by 3 by 1 mm., tipped with silver. There is a bend in the rod of the instrument in the petiole extending from the base of the leaf. The bend is a rounded angle of 90 degrees and extends for about 1 cm. from the base of the leaf. The blunt tip of the leaf is used from the outer surface of the eye and is nonpenetrating. This tip comes into action during the first stage of the operation and is used to impart delicate fish-angling jerks to the 6 o'clock border of the equator of the lens-in-capsule.[30] These fish-angling jerks are imparted in a forward and upward direction away from the vitreous. The tip of the dislocator

hitches and imparts these delicate jerks to the posterior lower border of the equator of the lens-in-capsule at 6 o'clock from the outer surface of the sclera, till a breach of about 2 mm. is effected in the zonule at 6 o'clock.

The tip with the adjoining portion of the leaf-shaped span comes into action during the second stage of the operation (fig. 1). The tip with the contiguous portion of the leaf-shaped span is insinuated into the breach of the zonule and below the lower border of the equator of the lens-in-capsule, padded by the cornea. The tip and span are used to give steady forward and upward tilting effort. The lower border of the lens-in-capsule is thus slowly and steadily rotated forward and upward, while the resistance of the attachment of the zonule gives way by lever action gradually from below upward.[31] After the lower border of the lens-in-capsule has rotated forward and upward up to 45 degrees, it clears itself out of the attached lower half of the cornea, and the second stage of the operation ends.

The bend of the mango leaf dislocator comes into action during the third stage of the operation. The hyaloid fulcrum acts as a socket fulcrum during this stage to accommodate the anterior surface of the lens-in-capsule within the hyaloid socket.[32] The bend of the mango leaf dislocator is hooked below and behind the posterior surface of the lens-in-capsule, while the anterior surface is socketed into the hyaloid fulcrum. By a final delicate turning movement of the mango leaf dislocator forward and upward, the detachment of the attached upper half of the zonule from below upward to 12 o'clock is completed without much effort. The advantage afforded by lever action is great during the third stage. The lens-in-capsule is therefore delivered without any appreciable effort.

But, in order that the lens-in-capsule may be rotated and delivered without any obstruction, it is necessary that the incision in the sclerocornea be sufficient for the passage of lens. Some surgeons recommend that the incision extend over two fifths of the corneal circumference, while others recommend that it extend over three fifths.

The safe delivery of the lens-in-capsule depends on proper limbic section, which must be sufficiently large to allow for the passage of the lens-in-capsule. Also the proper nutrition of the limbic flap after operation depends on proper limbic section, which should not be inordinately large. If the incision is too large, delivery will be easy, but the cornea will be hazy due to malnutrition. What is the proper corneal section, then, that meets both requirements?

It is a geometric fact that the diameter at 180 degrees is the broadest part of a circle. The equator of the lens-in-capsule is circular. The

socket within the ciliary body into which the equator of the lens-in-capsule is accurately socketed is also circular. But, the cornea fits on the sclerotic like the crystal of a watch. Therefore, the circumference at the base of the cornea is just a little narrower than the equator of the lens-in-capsule. In order that the largest diameter of the lens-in-capsule may safely be delivered, the points of puncture and counterpuncture should be chosen a little outside the limbic arc at its broadest diameter at 180 degrees. The point of the cataract knife on entry may pass through a bit of conjunctiva before piercing the coats of the eye, just a little outside the sclerocorneal junction; and again the counterpuncture should be just a little outside the sclerocorneal junction, emerging with a bit of conjunctival flap. This will insure a broad base with a bit of conjunctival flap, and at the same time the incision will not be too near the filtration angle and the ciliary body. After emergence at the counterpuncture, the knife should be steered to the line of the limbic arc and so also at the puncture, when the incision on that side is begun.

In this connection, the views expressed in 1868 by McNamara,[33] professor at Calcutta Medical College, about making the flap for intracapsular extraction are worth quoting: "In making the flap we must keep slightly external to the margin of the cornea so as to leave as large an opening as possible through which the lens may escape, its bulk when contained within the capsule being considerable."

Unlike capsulotomy, the whole of the lens-in-capsule is extracted with the lever action intracapsular operation. Therefore, an incision extending over the upper half of the circumference of the limbus with a broad base will make an outlet which will allow the lens-in-capsule to pass during its delivery without risk of rupture of the capsule or without undue application of force or violence. At the same time, there is no risk of the corneal flap suffering from malnutrition.

J. W. Wright [2] advised that the section be made one third of the corneal circumference, and Barraquer [26e] stated that it should be made two fifths of the sclerocorneal circumference with a small-tongued conjunctival flap at its middle third. The lens-in-capsule can be delivered through these passages, which are slightly less than half the corneal circumference, but only if the two following conditions are satisfied. It has been mathematically proved that an incision including half the circumference of the limbic arc plus a broader base is necessary for the passage of the broadest diameter of the equator of the lens-in-capsule. It is therefore necessary for the safe delivery of the lens-in-capsule through a passage of one-third or two-fifths the circumference

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33. McNamara, N. C.: Lectures on Diseases of the Eye, London, J. Churchill & Sons, 1868.

of the limbic arc that (1) the passage should dilate sufficiently or (2) the lens-in-capsule should undergo necessary molding. It may be that both dilatation of the passage and molding of the lens-in-capsule may go on hand in hand in the process of delivery. But there are cases in which with every effort delivery cannot be effected through small passages. In such cases the base of the incision has to be enlarged to effect safe delivery. The reason is that the cornea of senile persons is more or less sclerosed and does not as a rule dilate much. Moreover, a very hard cataract with a large lens and little cortical matter does not admit of much molding. To try to force a delivery under the foregoing conditions through a passage less than half the circumference of the limbic arc will necessitate much violence with the attendant risks. Again, in soft cataracts with thin capsules, such as the morgagnian or the intumescent variety, the thin capsules are liable to burst when undergoing excessive molding. It is better to take no risk but to make the incision half the circumference of the limbic arc at the outset and thus insure nonviolence and safe delivery of the lens-in-capsule.

On the other hand, if the section is made three fifths of the circumference of the limbic arc then so far as the nutrition of the corneal flap is concerned one is on the borderline of safety. For the first few days after operation the incised corneal flap depends for its nutrition on the supply of lymph that reaches it through the lymph channels of the attached portion of the cornea. Thus, if the incision is larger than three fifths of the circumference of the limbic arc the nutrition is insufficient. If the incision is just three fifths of the circumference, the nutriment that reaches the flap may be sufficient in healthy persons. But when one considers that for safe delivery of the lens-in-capsule an incision larger than one half of the circumference of the limbic arc is unnecessary, why then go to the extreme limit of safety, i. e., why make an incision three fifths of the circumference? In case of a bad cornea in a senile and debilitated patient there is risk of a corneal opacity with a flap even three fifths of the circumference of the limbic arc. Why take such an unnecessary risk?

Thus, with corneal flaps less than half the circumference of the limbus, as recommended by J. W. Wright [2] and by Barraquer,[26e] there is greater chance of a supply of nutriment to the incised corneal flaps; however this little advantage is more than counterbalanced by the risk of traumatic delivery in cases of hard cataract with large nuclei and by the risk of rupture of the thin capsules in cases of soft cataract. It is far better to admit the mathematical truth by making the corneal flap half the circumference of the limbus; delivery of the lens-in-capsule with nonviolence and without obstruction at the outlet is thereby insured.

It is better to include small bits of conjunctiva at the puncture and at the counterpuncture. Again, a small conjunctival flap at the termination at 12 o'clock is also useful. A thin layer of subconjunctival tissue underneath the conjunctival flap will prevent it from drying up like parchment and insure its agglutination with the episcleral raw cut within six hours after operation. These measures will open up new channels for the nutrition of the corneal flap after three days and keep the margins of the wound in the apposition necessary for healing by primary intention. Special dietetic measures for insuring nutrition of the corneal flap will be discussed in a future article. In view of all the foregoing facts, an incision in the limbus extending over the upper half of the sclerocornea, terminating in a small conjunctival flap at 12 o'clock and having a broad base covered with small bits of conjunctiva attached thereto, is advisedly made in the lever action intracapsular method, which involves no risks.

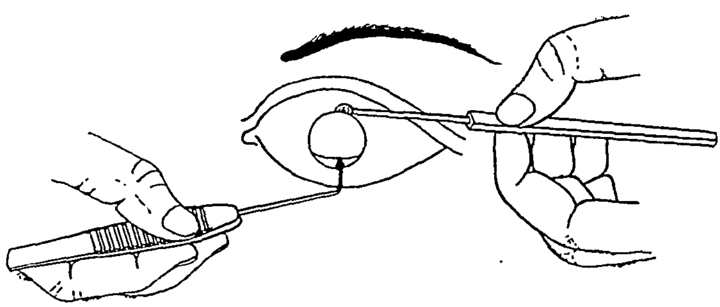

Fig. 1.—Diagram illustrating stage I and stage II of the operation. The hyalonavicular fulcrum is held by the unused hand with the thumb on one side and the other four fingers on the other side of the handle. It is applied horizontally and tangentially to the optic globe, socketing the upper equatorial border within the navicular fulcrum.

The mango leaf dislocator is held perpendicularly by the best hand with the thumb on the side of the handle continuous with the concavity of the band and the index and middle fingers on the side of the convexity. The tip and concavity of the mango leaf point forward and upward.

STAGE I: DISLOCATION OF THE LENS IN ITS CAPSULE

After the incision is made in the limbus, the lens-in-capsule is delivered by lever action. This is done in three stages. The first stage consists of dislocation of the lens-in-capsule.[30] In this stage, the attachment of the zonule is to be breached over a small area about 2 mm. near 6 o'clock. To perform this, the navicular socket of the hyalonavicular fulcrum is fixed at 12 o'clock, engaging the upper border of the equator of the lens-in-capsule, padded by the sclera. For operation on either eye the hyalonavicular fulcrum is held in the left hand of a right-handed surgeon (fig. 1) and in the right hand of a left-handed

surgeon with the thumb on one side of the handle and the remaining four fingers on the other side. The operator should change his position, standing where it is most convenient according to the eye on which he is operating. The upper border of the equator of the lens-in-capsule at 12 o'clock is just behind the attachment of the iris, which is visible through the incision at 12 o'clock. There is therefore no difficulty in finding it and socketing it into the navicular fulcrum.

The mango leaf dislocator is held in the right hand of a right-handed surgeon with the thumb on one side of the handle and the index and middle fingers on the other side. The hold of the thumb should be on the side of the handle which is continuous with the concavity of the bend of the rod, or the petiolar portion of the leaf. The index and middle fingers should be placed on the handle opposite to the thumb; they are placed, therefore, on the side of the handle which is continuous with the convexity of the bend, or the petiolar portion of the leaf. The instrument is held in the left hand in case the surgeon is left handed. The best hand is to be taken advantage of in the act of giving the least jerky efforts with the tip of the mango leaf dislocator to the lower and posterior border of the equator of the lens-in-capsule at 6 o'clock. Ambidexterity need not be acquired. The surgeon is at liberty to change his position of standing as required. He may take his stand at the head of the table (fig. 1) or at the right side or at the left side of the patient as is convenient.

To locate the position of the equator of the encapsuled lens at 6 o'clock from the outer side of the sclera, the tip of the mango leaf dislocator may be passed over the surface of the sclera from behind forward toward the limbus at 6 o'clock. At the line of attachment of the equator, which is generally about 3 mm. outside the limbic arc, some resistance is felt by the tip of the dislocator. However, it does not matter much if the curve of the attachment of the equator deviates a little to this or that side of the usual place, or even if its resistance may not be well felt by the surgeon. The tip of the mango leaf dislocator automatically hitches the lower border of the equator of the lens-in-capsule, whether it is at 3 mm. outside the limbus or a little anteriorly or posteriorly. This is because the fish-angling jerks are given from behind forward and below upward over the surface of the sclera toward the limbus at 6 o'clock. There is nothing else there to offer resistance to the tip of the mango leaf dislocator except the border of the equator of the encapsuled lens. Therefore, the tip of the dislocator cannot help but hitch the lower border of the equator when the operator jerks it at about its attachment. The only thing that is imperatively necessary is to give delicate jerks. If violent jerks are given, the encapsuled cataractous lens will be violently shot out; because lever action enhances the force or effort originally applied, forceful jerks act more violently.

What is wanted is only a small breach of about 2 mm. in the zonule at 6 o'clock. For this purpose, delicate jerks only are required. Since the operation is to be nonviolent, how can the violent jerks be avoided? The delicacy of the jerks can be insured if the flexor muscles of the index and middle fingers only are used in giving the jerks and not the muscles of the hand. These delicate jerks should be repeated until the desired breach in the zonule is effected. Then the second stage of rotation begins.

The number of fish-angling jerks required for making a small breach in the zonule is variable, because the strength of the anchorage of the zonule is variable. For the extraction of a morgagnian cataract a single delicate jerk is sufficient. This is because the zonular attachment is extremely weak, so much so that it is said to become dislocated by an angry look. On the other hand, in the case of disciform cataract, half a dozen or more delicate jerks are required, because the anchorage is very firm. However, it matters little whether one or two jerks or a dozen or more are used, so long as the jerks are nonviolent. Ultimately, the zonular attachment cannot but give way to the required extent at 6 o'clock. As these jerks are given in a direction away from the vitreous, there is no possibility of rupturing the hyaloid membrane or of expressing the vitreous. The direction of the application of the effort or power is just the opposite of that used in Smith's [3] pressure method. The nature of the effort is also different, and the scientific principle involved is fundamentally different. With Smith's pressure method the direction of the application of power for the extraction of a hard cataract is directly backward toward the optic nerve. For the extraction of a soft cataract, the application of power is downward toward the patient's feet. The pressure is continuous and intended to raise the tension of the vitreous gel, to deform the vitreous and thereby to rupture the zonule. On the other hand, with the lever action intracapsular method, irrespective of whether the cataract is soft or hard, the efforts made are jerky and superficial; neither continuous pressure nor deep, indeed, no pressure, is given. For both hard and soft cataracts, the efforts made are from behind forward in a direction away from the vitreous and from below upward, intended to hitch the lower border of the equator of the lens-in-capsule upward and forward away from its bed on the hyaloid fossa without disturbing the hyaloid membrane. The surgeon is therefore at liberty in extracting both hard and soft cataracts to repeat his delicate fish-angling jerks without hesitation and without fear of loss of vitreous until a small breach is made in the zonule at 6 o'clock. As no pressure or suction is applied either on the vitreous or on the lens-in-capsule, the question of expression or suction of the vitreous does not arise. The only thing required for the surgeon is a little patience for a few seconds until a breach is made in the zonule.

While nonviolence on the part of the surgeon need not be often hinted at and while the technic of the lever action intracapsular method is nonviolent in its very essence, still there is risk of violence from an unexpected quarter. This is from the patient. At any stage of the operation, from a trivial cause or from no cause, the patient may suddenly become impatient and nervous. He may squeeze the orbicularis muscle so violently that a considerable quantity of vitreous may be squeezed out. This violence by the patient is unintentional. It is no use remonstrating with him, as that makes him more nervous and more uncontrollable. As this sort of violence on the part of the patient at any stage of the operation is disastrous and as it will do no good to warn him beforehand against such sudden nervousness, one should take every precaution to control the orbicularis muscle and guard against its screwing up.

In preanesthetic days, control of the orbicularis muscle by a trained assistant was imperatively necessary for the success of the cataract operation. Nowadays, the eye speculum, the eyelid retractors, facial akinesia, retrobulbar anesthesia and external canthotomy have all contributed to keep the orbicularis muscle under control and to paralyze it.

In modern times, Smith,[3] after trying all the aforementioned methods and a few more, stated that it is not safe to dispense with the services of a competent assistant who is especially trained. With the lever action intracapsular method also the assistant should be competent enough to control the orbicularis muscle promptly by the use of the stirrup eyelid retractor (fig. 2) and also by his fingers on the superior orbicularis muscle in case of emergency if the patient suddenly squeezes the lids. At any rate, this sudden violence, which is disastrous, must always be guarded against.

STAGE II: ROTATION OF THE LENS-IN-CAPSULE

For the second stage of the operation the hyalonavicular fulcrum should be held between the thumb on one side of the handle and the other four fingers of the unused hand on the other side (fig. 1). The instrument should be applied horizontally and tangentially to the optic globe, as in the first stage. The navicular socket is to be kept fixed at 12 o'clock as before, socketing the upper border of the equator of the lens-in-capsule into its concavity, padded by the sclera.

The mango leaf dislocator should be held between the thumb on the side of the handle which is continuous with the concavity of the bend of the rod, or the petiole, and the index and middle fingers on the side of the handle which is continuous with the convexity of the bend of the rod, or the petiolar portion of the leaf (fig. 1). The instrument is to be held by the best hand of the surgeon, as before, and is to be

held perpendicularly to the optic globe with the concavity of the rod forward. The tip of the leaf should point upward. The tip of the dislocator and the adjoining portion of the leaf span should be insinuated into the breach in the zonule at 6 o'clock. Thus, the tip with the leaf span is slipped below and behind the lower border of the lens-in-capsule as a wedge, the cornea intervening. The lower or inferior border of the cataractous lens-in-capsule, rendered into a more or less rigid lever, is thus slowly tilted forward and upward away from its bed in the hyaloid fossa of the vitreous on the navicular fulcrum fixed to the upper or superior border of the equator of the lens-in-capsule, like the lid of a teapot or jug on its hinge. The slow tilting upward and forward movement of the lower border of the lens-in-capsule are effected by steady delicate effort of the leaf span from below upward and behind forward, away from the vitreous.[31] Only delicate efforts are required, because a lever of class II is formed in the mechanism of lever action. This is because the resistance of the zonular attachment is placed between the navicular fulcrum, socketing the upper 12 o'clock border of the more or less rigid cataractous lens-in-capsule and forming the rod of

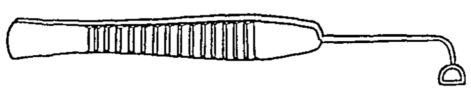

Fig. 2.—Dutt's stirrup eyelid retractor.

the lever, and the effort applied to the diametrically opposite end of the cataractous lever by the leaf span of the mango leaf dislocator at 6 o'clock. A lever of class II always acts as a mechanical advantage, because in this case $\frac{\text{arm of effort}}{\text{arm of resistance}}$ is greater than unity. As more and more of the zonular attachment gives way during the upward and forward rotation of the lower border of the lens-in-capsule, more and more of it gives way from below upward, and thereby the point of resistance of the zonular attachment approaches nearer and nearer the fulcrum fixed at 12 o'clock. Or, in other words, as more and more of the zonular resistance is peeled off from below upward and the point of resistance approaches nearer and nearer the fulcrum, the denominator of the foregoing equation, viz., the arm of resistance, becomes lesser and lesser; while the numerator, viz., the arm of effort, i. e., the perpendicular distance from the point of application of the effort at 6 o'clock to the point of application of the navicular fulcrum at 12 o'clock, remains constant. Hence, the resulting mechanical advantage derived from lever action becomes greater and greater till the lower border of the equator of the lens-in-capsule has rotated on the navicular fulcrum at 12 o'clock for about 45 degrees. Thus, the mechanical advantage gained by lever action throughout the second stage of rotation of the

lens-in-capsule becomes progressively greater and greater. The second stage ends when the lower border of the lens-in-capsule has rotated clear out of the attached portion of the cornea.

It will be interesting and important to discuss the physical and anatomic relations between the vitreous and the lens-in-capsule and the possible disturbance of the vitreous owing to the dislocation and rotation of the lens-in-capsule and to the detachment of the zonule. The lens-in-capsule is nicely set in its bed in the hyaloid fossa of the vitreous, like a boat set in a muddy bank or dock anchored all round. The posterior surface with the posterior pole of the lens-in-capsule is just in apposition with the vitreous in the hyaloid fossa, which constitutes the hyaloid membrane. All round its equatorial border the lens-in-capsule is anchored by the zonule of Zinn in bundles of three rows. Thus, there exist all the physical conditions necessary for functioning of capillary attraction between the convex posterior surface of the lens-in-capsule and its concave bed in the hyaloid fossa. In order to avoid the powerful resistance of the capillary attraction, it is necessary either (1) that the lens-in-capsule be glided over the hyaloid fossa, as a boat set in a muddy bank or muddy dock is glided or (2) that a border of the lens-in-capsule be tactfully raised like the lid of a jug or teapot on its hinge by lever action, so as to admit atmospheric pressure between the hyaloid fossa and the posterior surface of the lens-in-capsule. In order that (1) a gliding movement of the lens-in-capsule may be possible, first, the whole of the zonular attachment must be detached previously; otherwise the lens-in-capsule cannot be glided out of its setting. Second, complete iridectomy should be performed previously so as to give clear passage to the lens-in-capsule. If iridectomy is not performed or only a buttonhole iridectomy is done, the foremost upper edge of the advancing lens-in-capsule is tucked into the upper angle of the posterior chamber and is netted by the attached iris. In order that (2) a lid on hinge lever action may be possible, it is not necessary to perform iridectomy. It is only necessary that a portion of the zonule attached to that border should be previously breached, so that there may be a passage for the atmospheric pressure to act between the posterior surface of the lens-in-capsule and the bed in the hyaloid fossa. It is therefore obvious that this second mechanism, i. e., the lid on hinge mechanism, is better than the first, i. e., the gliding-boat mechanism. The lid on hinge mechanism is exactly what is produced in the lever action intracapsular method.[31] A small breach is first made in the zonule at 6 o'clock by fish-angling jerks applied from the outside so as to admit atmospheric pressure through the breach. Then, the extreme lowest border of the equator of the lens-in-capsule at 6 o'clock is slowly and gradually raised like a lid on its hinge by lever action with the effort applied

from the leaf-span of the mango leaf dislocator on the navicular socket fulcrum. Thus, atmospheric pressure is admitted into and acts between the posterior surface behind the lowermost border of the lens-in-capsule and its corresponding bed in the hyaloid fossa. By this lid on its hinge mechanism, the capillary action and cohesion between the posterior surface of the lens-in-capsule and its bed in the hyaloid fossa are avoided. Moreover, as more and more of the posterior surface is raised from its bed on the hyaloid fossa from below upward, at the same time more and more of the zonular attachment is peeled off from below upward by one and the same lever action mechanism during rotation of the lens-in-capsule.

The illuminating notes in this connection and the diagrams given by Barraquer [26c] are worth noting: "There are those who believe that the posterior surface of the convex lens is in intimate contact with all points of its surface with the anterior surface of the vitreous from the identical concavity and the perfect juxtaposition of the two surfaces producing an adherence that renders it impossible to extract the lens by drawing it directly forward, as it would be impossible to separate the two surfaces thereby." The direct forward suction or traction and the impossibility of separating the two surfaces thereby are illustrated in figure 3 *A*. The possibility of success by gliding action in effecting upright delivery of the lens-in-capsule with upper edge foremost is illustrated in figure 3 *B* The arrows indicate the direction of the application of power.

Barraquer did not express his personal opinion about the necessity of avoiding capillary action and adherence of the posterior surface of the lens-in-capsule and its bed in the hyaloid fossa. But action is more eloquent than words, as the practical is more eloquent than the theoretic. What is done in Barraquer's suction method and also in the forceps traction methods to attain success?

Barraquer [26c] gave the following directions for effecting upright delivery of the lens-in-capsule with the upper edge foremost. "Once having caught hold of the crystalline lens by the force of the vacuum, and torn the fibres of the zonula, in order to withdraw the cataract from its position, if an iridectomy has been made it is sufficient to raise its superior border whilst it works upward, in such a way that the inferior border of the lens follows the curve of the patellar fossa, in contact with it, without exerting the least pressure or producing friction on its posterior surface." Thus, it is necessary that complete iridectomy be done and that the fibers of the zonule be completely detached in order to withdraw the cataract from its position by gliding over the curve of the patellar fossa in contact with it and to deliver the upper edge foremost.

Barraquer omitted to illustrate by diagram the mechanism of tumbling, though he called it "my usual method," nor did he explain how skilfully and scientifically capillary action is avoided thereby. Although delivery of the lens-in-capsule by tumbling is effected with Smith's pressure method as well as with the Knapp[11]-Török[17]-Elschnig[16] forceps traction with pressure, no one has so far explained the mechanism by which atmospheric pressure is admitted and capillary action avoided. How far the theory of "lid on its hinge" is applied in actual practice and also in "lid tumbling" will be considered.

In tumbling, which Barraquer[26e] called "my usual method," simple extraction with a peripheral buttonhole is made. The suction cup should hold the lens-in-capsule a little lower than the center of the pupil and behind the iris. Then the inferior border of the cataract is raised from its bed on the patellar fossa and its zonula is ruptured and tumbled

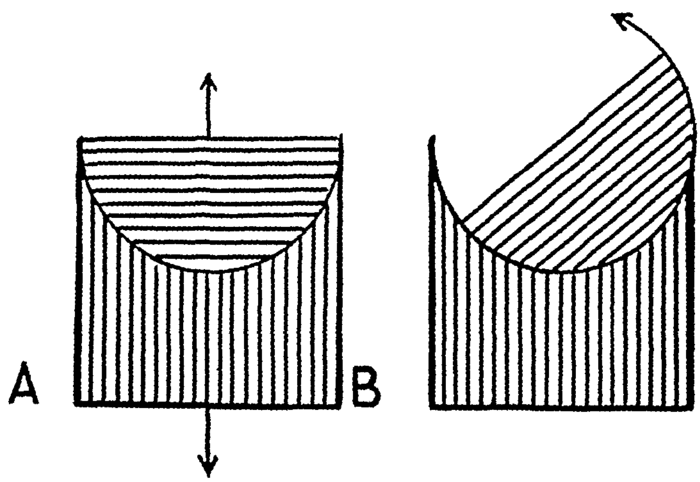

Fig. 3.—*A,* diagram illustrating direct forward traction or suction. *B,* diagram illustrating gliding boat. (Redrawn after Barraquer.[26e])

upside down, its inferior border thus rubbing the posterior surface of the iris. The delivery is effected with the lower edge foremost.

With the Knapp-Török-Elschnig[16] method, a small slit in the iris is made with de Wecker's scissors at about its root. Then the capsule is seized with the capsule forceps as low down as possible below the dilated pupil. Then the lens is gently rocked from side to side to breach the zonule. Thereafter, the lens-in-capsule is raised in the forceps, so that its lower edge is brought forward and upward through the pupil and delivered with the lower edge foremost, as in tumbling, the lens hook aiding the maneuver with pressure from the outside.

It is therefore clear that with Barraquer's[26] method a peripheral buttonhole is made in the iris, and with the Knapp-Török-Elschnig[16] method a slit is made near the root of the iris, but with neither is complete iridectomy done. A hold by the suction cup or by the capsule forceps is secured on the lens-in-capsule as low as possible. The zonular

attachment of the lower portion is then breached. The lower edge of the lens-in-capsule is raised either with the suction cup or with the capsule forceps from its bed on the hyaloid fossa and gradually tumbled. The zonular attachment, therefore, slowly gives way from below upward. Thus atmospheric pressure is brought into action, and capillary action and cohesion between the posterior surface of the lens-in-capsule and the hyaloid fossa cannot take place.

The lower the hold on the lens-in-capsule for the application of power to raise its lower border at 6 o'clock from the hyaloid fossa, the greater the advantage. With the lever action intracapsular method, the lowermost border of the lens-in-capsule is the strategic point on which the power or effort is applied from the outside. With neither the suction nor the traction method can the hold on the lens capsule for the application of power by the suction cup or the capsule forceps be made much lower than the lower border of the pupil because of the iris and because of the narrow nook of the posterior chamber below, where no instrument can reach.

The direction of the application of power by suction or by traction or by lever action is uniformly away from the vitreous and at the same time forward and upward, intended to raise the lower border of the lens-in-capsule from the hyaloid fossa of the vitreous and also to rotate it by tumbling or by turning on a navicular fulcrum. This mechanism is found practically convenient and successful in avoiding capillary action and cohesion between the posterior surface of the lens-in-capsule and its bed on the hyaloid fossa of the vitreous.

STAGE III: DETACHMENT OF THE ZONULE

When by rotation on the navicular fulcrum the lower border of the lens-in-capsule has rotated upward and forward away from its bed on the hyaloid fossa till it has cleared itself out of the attached lower half of the cornea, the stage III of detachment of the zonule begins.[32]

The hyalonavicular fulcrum is turned between the thumb and the fingers of the hand not used, in which it has been held throughout the operation, so as to bring the hyaloid socket fulcrum into action. The hyaloid fulcrum is placed horizontally and above the anterior surface of the lens-in-capsule engaging the convexity of the anterior pole of the lens-in-capsule in the concavity of the hyaloid fulcrum. The anterior pole and anterior surface of the encapsuled lens are now in direct contact and within the hyaloid socket fulcrum. The convexity of the anterior pole of the lens-in-capsule is thus securely socketed within the concavity of the hyaloid fulcrum, thereby forming a ball and socket joint fulcrum, and cannot slip. The mango leaf dislocator is now held horizontally and tangentially to the optic globe between the thumb and the index and middle fingers of the best hand of the surgeon. The

concavity of the bend of the petiolar portion of the rod of the mango leaf dislocator is placed in such a way that it hooks around the lower and posterior border of the encapsuled lens. As the lower border of the lens-in-capsule has now freed itself from the attached lower portion of the cornea, the concavity of the bend of the mango leaf dislocator is in direct contact with the posterior surface of the lower border of the encapsulated lens, nothing intervening.

By a delicate turning forward effort of the mango leaf dislocator, the lower border of the cataractous lens-in-capsule is made to turn forward and upward on the hyaloid socket fulcrum. Now that the points of resistance on both sides in the attachment of the zonule have approached much nearer to the fulcrum than before, the mechanical advantage gained by lever action has become much greater. Therefore, by a slow, steady and delicate turning effort with the advantage of lever action, the attachment of the remaining portion of the zonule of the lens-in-capsule is gradually detached completely from below upward without any appreciable resistance up to 12 o'clock. The encapsuled lens is held between the bend of the mango leaf dislocator and the hyaloid socket fulcrum. The encapsulated cataractous lens is nonviolently delivered by the temporal side of the incision.

COMMENT

The adaptation and application of the scientific principle of lever action to surgical instruments are not new or recent. Indeed, surgical scissors, elevators and forceps of various designs have been in use ever since the Susrutian age, 1,000 B. C. The ancients were enthusiastic about lever action and were aware of its unlimited potentiality. Archimedes (300 B. C.) once exclaimed: "Give me a fulcrum and I can rotate the earth!" Well, here is the hyalonavicular fulcrum. No wonder that a modern Archimedes can now rotate the most stubborn lens-in-capsule with perfect nonviolence.

A NEW METHOD FOR TRANSPLANTING PTERYGIUM

EDWIN M. NEHER, M.D.
SALT LAKE CITY

Ophthalmologists have recognized for many centuries that the only treatment for true pterygium is surgical intervention. Many and various operations have been devised for the removal of the pterygium, either some form of excision or transplantation in the lower cul-de-sac being used. These operations are well known to all ophthalmologists.

Transplantation of the entire pterygium beneath the upper bulbar conjunctiva has never been described. It has much merit and possesses advantages over all other operative methods.

OPERATIVE TECHNIC

After the usual surgical preparation of the conjunctival sac is carried out and local anesthesia is obtained, the lids are held apart with an eye speculum. The following steps are then performed:

The neck of the pterygium, including the margins, is grasped with a strong, narrow tissue forceps. While gentle traction is exerted upward with the forceps, a sharp von Graefe knife is used to dissect carefully and accurately the head and neck from the cornea and limbus, as in *A* of the illustration.

With the pterygium still held with the forceps, a pair of small curved scissors is used to make an incision approximately 1 cm. long, beginning at the upper end of the incision at the limbus and forming an angle of about 30 degrees with the upper margin of the pterygium, as in *B*. Then with the concave surface facing the eyeball, the closed scissors are inserted through the incision, and the blades are opened gently to free the pterygium and bulbar conjunctiva from the underlying sclera as far as the lower cul-de-sac.

The hold on the pterygium is released, and the upper conjunctival margin of the incision is picked up and the bulbar conjunctiva is freed from the sclera with a small strabismus hook, thus preparing a place into which the head of the pterygium is soon to be transplanted, as in *C*.

Each curved needle of a double-armed black silk suture is passed through the head of the pterygium from within outward, there being sufficient tissue between the two ends of the suture to make a firm and secure hold, as in *D*.

The speculum is removed, and the upper lid is retracted with a Desmarres lid retractor, as in *E*. With the eyes in their primary position, the head of the pterygium is grasped and used as a pattern, being placed on the upper bulbar conjunctiva in such a position as to cover the denuded area of the sclera completely and to make perfect coaptation of the normal conjunctiva at the limbus. The

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Read at the meeting of the American Ophthalmological Society, San Francisco, June 10, 1938.

position of the pterygium is mentally noted, or a better plan is to mark the location of the head on the conjunctiva with aniline stain.

The needles on the double-armed suture are carried through the incision and beneath the conjunctiva, and are brought out through the spot of aniline stain, as in *F;* tension on the suture slips the head into proper position, and tying the ends holds it there.

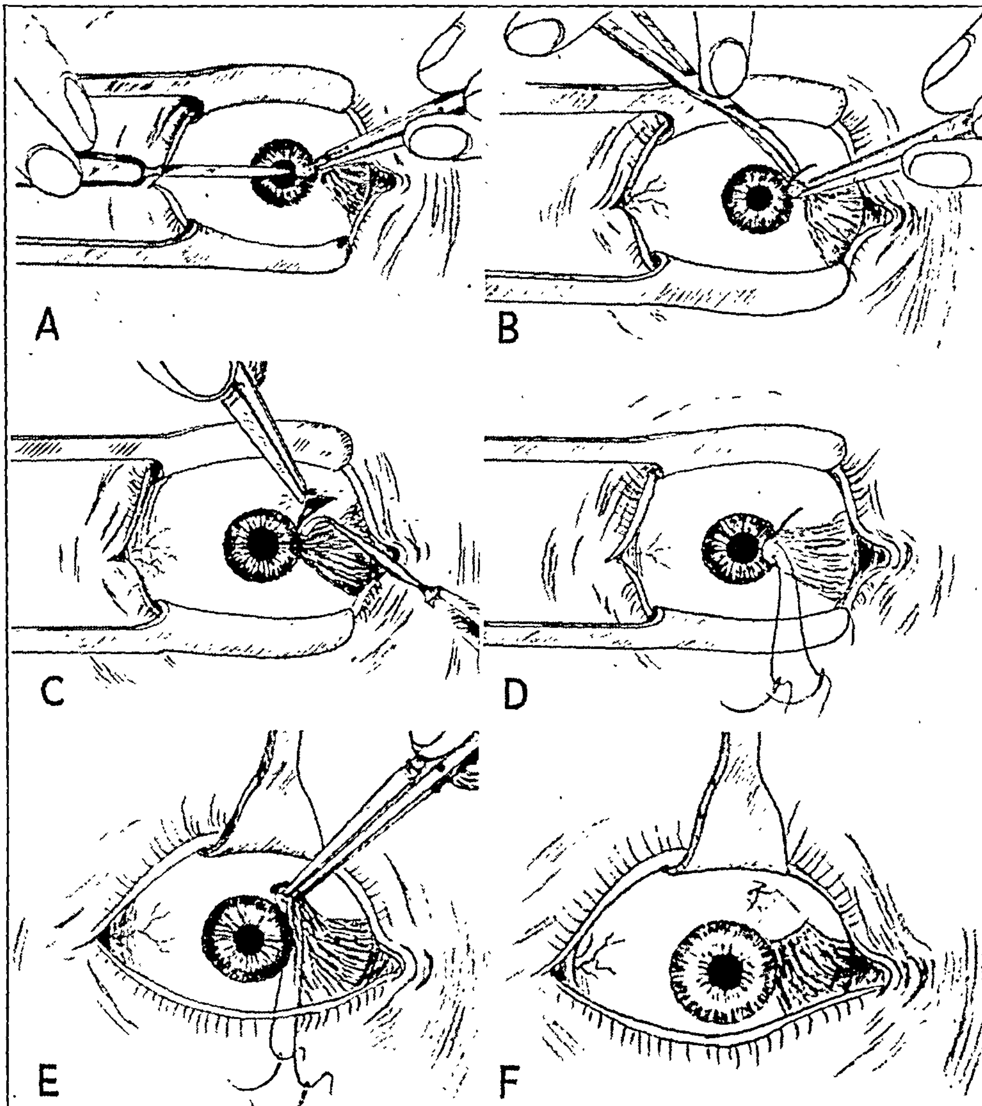

Fig. 1.—Technic of the operative procedure.

Thus the pterygium is transplanted beneath the upper bulbar conjunctiva, and the denuded area of the sclera at the former site of the pterygium is now completely covered with normal conjunctiva.

Atropine should be applied and both eyes should be bandaged for one day; the eye that was operated on should be bandaged for two or three days longer.

COMMENT

By removing the pterygium from the cornea, as depicted in *A* of the illustration, the operator can see the field of operation and control the depth of the dissection. Cutting the cornea deeper than necessary

is as bad as not completely removing all of the pterygium, since each delays the process of healing and encourages the formation of recurrent vascular tissue on the cornea.

It is important to make the incision in the bulbar conjunctiva before any attempt is made to free the pterygium and conjunctiva below from the underlying sclera, since this gives greater freedom for the manipulation and there is less likelihood of tearing the conjunctiva.

Should the pterygium be large, thick and fleshy or contain sclerotic areas, these should be dissected from the overlying conjunctiva. Their removal lessens the amount of congestion and shortens the period of healing.

A small strabismus hook or a spatula is useful to separate the upper bulbar conjunctiva from the underlying sclera.

The retraction of the upper lid with a Desmarres retractor permits a better view to place the "pattern" on the bulbar conjunctiva correctly. It also provides greater space to insert and tie the sutures.

It is not necessary to place the head of the pterygium as far as the bottom of the upper cul-de-sac; it should be carried only far enough beneath the bulbar conjunctiva to make accurate juxtaposition of the normal conjunctiva at the limbus, without undue tension of the tissues.

If both eyes are rather firmly bandaged for one day, the normal conjunctiva will adhere to the denuded sclera in the proper position. The sutures may be removed on the third day.

While the advantages of transplanting the pterygium beneath the upper bulbar conjunctiva are many and obvious, a few of them are noted:

The site of the former pterygium is completely covered with normal conjunctiva the fibers of which take a vertical position at the limbus, making the return of the pterygium unlikely.

There is no break in the continuity of the normal conjunctiva covering the denuded sclera; hence, less probability of congestion or infection and return of vascular tissue on the cornea.

The incision in the conjunctiva and the pterygium are hidden beneath the upper lid, thus shortening the period of disability.

The normal pressure of the upper lid against the eyeball holds the tissue in position and protects the normal healing of the wound from irritation and disturbance.

The upper bulbar conjunctiva is constantly washed by sterile tears, which together with its upper location, minimizes the chances of infection.

The operation is easily performed, the accurate covering of the denuded sclera can be definitely planned and executed, and the final result is equally satisfactory to the surgeon and the patient.

Suite 1120 Boston Building.

DISCUSSION

DR. FRANK E. BURCH, St. Paul: For the reasons given by Dr. Neher I have been transplanting pterygium by this method for the last twenty-five years. I believe that many use this technic as being more logical than the one usually recommended by McReynolds.

DR. CONRAD BERENS, New York: If I understand Dr. Neher's technic, I have used a method similar to this procedure of transplanting pterygium upward (fig. 2). In 2 cases, the cornea was again invaded. In 1 case the invasion occurred after three years and in the other after eight years. Recently I have drawn the conjunctiva together between the pterygium and the cornea and turned the pterygium more sharply upward.

DR. E. C. ELLETT, Memphis, Tenn.: I prefer to remove the head of the pterygium by evulsion with a flat, blunt hook. If a knife is used for the purpose, it may go either too deep or not deep enough. In order to have a raw surface of the pterygium to apply to the raw surface of the conjunctiva, I scarify the upper surface of the pterygium

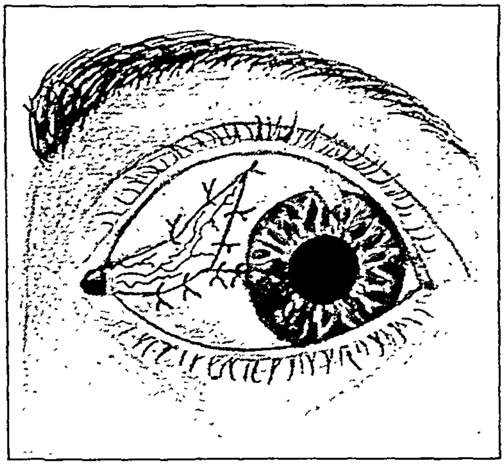

Fig. 2.—Transplant of pterygium upward (modification of Miller's technic of Desmarres' operation).

with a serrated chalazion curet. The suture is placed through the head of the pterygium and through the conjunctiva close to the cornea, in order to obviate leaving a small raw area along the edge of the cornea, which is a favorable site for the development of granulation tissue. When the suture is brought out through the conjunctiva it is threaded through two openings in a small silver suture plate; this avoids puckering up the tissues and keeps them spread out flat.

DR. EDWARD JACKSON, Denver: I have not tried Dr. Neher's method, but it seems to fulfil the indications for a successful operation better than any procedure I have tried. I learned a good many years ago that success apparently depends almost entirely on getting a close union at the corneal margin between the conjunctiva and the underlying tissues. This method of Dr. Neher, by which one would have a relatively thin edge of pterygium where it was drawn under the conjunctiva, and his incision, starting at the limbus, where the conjunctiva and the sclera are closely united normally, and running up and allowing room for the whole pterygium to be slipped under it, certainly

would afford an immediate, quick and close union of the conjunctival covering of the pterygium where it is drawn into the slit. Bringing the pterygium underneath the conjunctiva in that way would avoid the difficulty to which Dr. Berens alluded, which necessitated repetition of the operation.

Dr. Edwin M. Neher, Salt Lake City: I have searched the literature as thoroughly as I could but have found no description of this operation. Until such a description can be found, I assume that the operation can be considered as new.

As Dr. Jackson especially pointed out, the important thing is to have the conjunctiva joining on the limbus, not overlapping, but in accurate position to the limbus, with the fibers in the vertical position.

SOME PHYSIOLOGIC AND PHARMACOLOGIC REACTIONS OF ISOLATED IRIS MUSCLES

PARKER HEATH, M.D.

AND

C. W. GEITER, M.D.

DETROIT

This paper is a preliminary report of an experimental study of some reactions of isolated iris muscles, a new autographic method being used. Both physiologic and pharmacologic stimulators and depressors were employed. The delicate method showed physiologic tissue response and permitted pharmacologic instead of toxic reactions.

METHOD AND APPARATUS

The sections of iris were taken from the enucleated eyes of anesthetized or of stunned animals, mounted with clips and at once suspended in oxygenated Ringer's solution. The temperature was controlled (from 32.7 to 33.2 C.) and also the p_H (from 7.5 to 7.7, LaMotte colorimetric method). The suspension was fixed below and was attached above by a delicate thread to a galvanometer at a controllable tension. To increase the load of work, the muscles contracted against the elasticity of delicate coiled springs, and conversely a return to a relaxed state was favored by the stored energy in the springs. The load of work on each preparation was small. The average weight of an immersed suspension of clips and sphincter was 0.0507 Gm. The weight of the suspension and clips in the air was 0.0759 Gm. Muscle fibers of the suspension were comparatively parallel, an ideal physiologic preparation. The sections were started by incision with a cataract knife through the iris and were completed with iris scissors. The clips were curved to conform to the curve of the iris. Mechanical deflections of the galvanometer indicator, due to contraction and relaxation of the muscles, produced in a constant electromagnetic field differences in potential. These differences in electric current thus formed were conducted through amplifying tubes and rectifier to a string galvanometer. By deflection of a mirror on the string (as in electrocardiograph) a light beam recorded changes on a sensitized bromide paper. Time was electrically recorded. This arrangement, the electromyograph, was extremely sensitive to change in muscle tension, and did not cause undue work. By amplifications, minute changes could be recorded on the myogram. Also slight variations were recorded due to the time rate of the myograms—five seconds time made ⅜ inch (0.9) of record. Improved technic in removing the sectors has cut down the need for amplification except for recording minute changes. It now takes from three to five minutes to remove and mount the sections.

From the Ophthalmology Department of Wayne University and the Research Laboratory of Frederick Stearns & Co.

PHYSIOLOGIC RESPONSE AND SPONTANEOUS MOVEMENTS

Freeing the sphincter or dilator under Ringer's solution from its antagonist in the animal led to a contraction of the fibers. The suspended sphincter characteristically showed in outline the figure 8, and the dilator contracted markedly. This may have been due to the mechanical stimulation of cutting and mounting and probably was not due to freedom from tension of the opposing muscle. After from ten to twenty minutes in a bath of Ringer's solution, the preparation relaxed from the mechanical stimulation. A fresh preparation suspended with minimal tension showed a varying and uneven tonus. With minimal stimulation and a light work load, both dilator and sphincter showed an

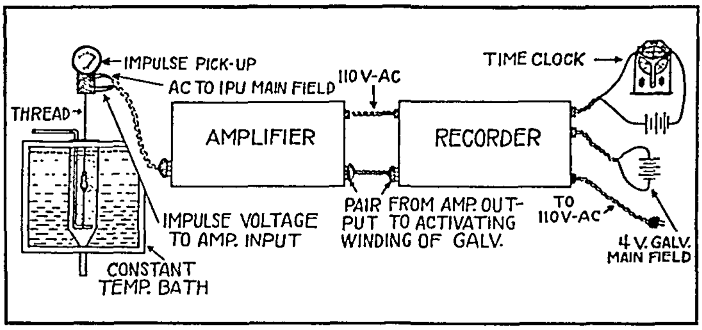

Fig. 1.—Outline of apparatus arrangement, showing the suspension in the bath, the impulse pick-up, the amplifier and rectifier and the recording apparatus, consisting of a galvanometer, mirror, light beam, timer and sensitized paper.

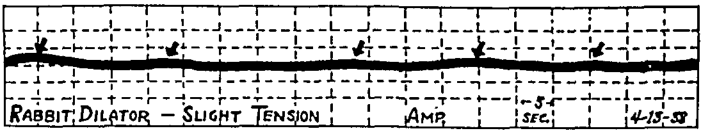

Fig. 2.—Rabbit dilator, fresh preparation; slight tension and amplification; irregular rhythm, from fifteen to twenty seconds. More tension would increase the response and lengthen the intervals; more amplification would show secondary curves.

irregular contracting and relaxing action (fig. 2). With heavy work and overload, the responses were gradual and the curves free from steps, the top showing a tetanic contraction. The curve of spontaneous contraction is made up of several parts: (1) an irregular steplike contraction phase, (2) a short plateau and (3) irregular relaxation phase with a decline to a no tension position and then a repeat to a lower rise, each rise and fall being associated with much variation. Some preparations showed rhythmic contraction, especially with the steadying effect of a light load, the time interval being about from twenty to sixty seconds. Our impressions were that the fibers of the iris muscle con-

tracted with some disassociation and asynergia. The maximum contraction or summation effect showed this. In other words, the peak responses were uneven and not level. The fatigability interval was short. After prolonged rest (about one hour), practically equal responses resulted from the same stimulus. A rebound from sudden changes in load increased the contraction responses (fig. 3). The total workability of the dilator sectors, contrary to usual reports, seemed to exceed that of the sphincter ring. In this we agree with Magitot.[1] We also agree with ten Cate,[2] who employed the Wessely[3] suspension method and recorded photographs of iris sectors, that there are spontaneous movements. But our sensitive methods in the rabbit suggested an irregular rhythm. Solely after noting the fatigability and relative strength of suspensions of isolated dilator and sphincter in vitro, we gained the impression that in vivo there cannot be actual antagonism between the two muscles any more than there is an active

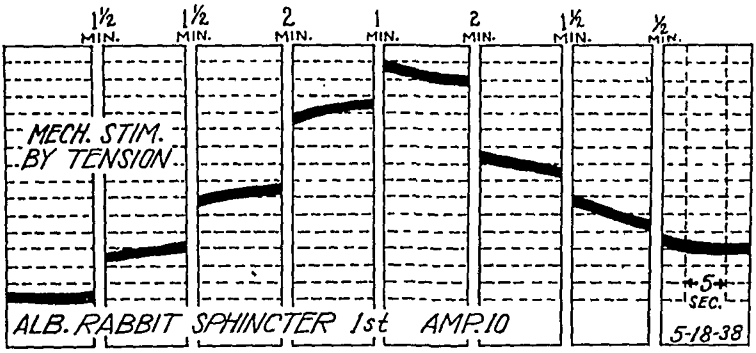

Fig. 3.—Albino rabbit sphincter; enucleated eye slightly sensitized in iced Ringer's solution for three hours; amplification, 10. The muscle was stimulated seven times by an increase of tension or work, the curve continually rising after such stimulus.

antagonism between the internal and the external rectus muscle. The notion that the sphincter is active and the dilator passive seems fallacious; also that the dilator acts by "blockade" and is not able to relax actively, except through the ability of a gliding blockade. In our opinion the dilator does not maintain a continuous tension, contrary to Poos.[4] Our suggestion is that there is a perfect cooperation between the sphincter and the dilator. The contraction of one is associated with the relaxation of the other. Both physiologic and pharmacologic response of sectors of isolated iris muscle were markedly

1. Magitot, A.: L'iris, étude physiologique sur la pupille et ses centres moteurs, Paris, Gaston Doin, 1921.
2. ten Cate, J.: Arch. néerl. de physiol. **6**:258, 1921-1922.
3. Wessely: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **42**:26, 1920.
4. Poos, F.: Arch. f. exper. Path. u. Pharmakol. **126**:307, 1927.

reduced in the presence of free blood. The older the stored preparation, the more sensitive, quicker and shorter the response. The response of iris muscles from eyes of old animals was low. Previous inflammatory disease of the iris, such as iritis, also sharply reduced the reactions. Pigmented and unpigmented irides responded equally. The muscles were sensitive to oxygen supply; increased reaction and rhythm followed the plus supply and decreased the minus supply. Lowering the temperature of the bath caused contraction, and rising temperatures caused relaxation. We worked in a fairly narrow temperature range (about 0.5 C.). The latent period responses of the smooth iris muscle of the rabbit were like those of other smooth muscles, estimated from one half to two seconds. This time variability seemed to be due to the effect of dilution. The pharmacologic responses to stimulators showed an interesting short, active depression phase before stimulation

Ion Reactions According to Poos *

Chemical Agent	Sphincter	Dilator	Comment
Potassium	Stimulator	Depressor	Increase tonus and spontaneous rhythm
Barium	Stimulator	Depressor	Strongest stimulator
Strontium	Stimulator	Depressor	Strong effect
Calcium	Depressor	Stimulator	
Magnesium	Depressor	Stimulator	Similar to epinephrine

* Poos worked out the effect of potassium, magnesium, calcium, strontium and barium ions, using neutral chloride salts in hundredth molar solution. With Ringer's solution isotonicity maintained by reducing the sodium chloride; the corresponding quantities of sodium chloride were determined after considering water of crystallization by interpolation from the depression of the freezing point. The solution contained, besides the salts to be examined, the correspondingly reduced quantity of sodium chloride and the other salts in Ringer's solution. No bicarbonate was added to the solutions of strontium and barium. Poos found that the deviation from normal Ringer's solution (p_H 7.5) was too small to be considered a source of error (p_H from 7.3 to 7.6).

began. The rabbit sphincter and dilator, like other smooth muscles, are sensitive to acid and alkali changes. An increase of hydrogen ion causes a decrease of tonus, and a decrease raises the level of tonus. We worked in a range of p_H from 7.5 to 7.7. Stimulators for both sphincter and dilator are cold, mechanical agents, electric current, oxygen plus and alkalization. Depressors for both sphincter and dilator, and especially the sphincter, are oxygen minus, heat, acidification, free blood, aged tissue, iritis aftermath, overwork, fatigue and trauma.

SOME PHARMACOLOGIC REACTIONS

The work begun by Loewi on the transference of nerve impulses to the cell by acetylcholine opened a new field in autonomic neurology and its pharmacology. This humoral or chemical mediation hypothesis is well, but not completely, developed. It seems that bound acetylcholine exists, that it may be produced by the preganglionic nerve endings (Feldberg and Vartiainen [5]), that it acts on the cell and that a choline

5. Feldberg, W., and Vartiainen, A.: J. Physiol. **83**:103, 1934.

esterase of unknown origin found in fluids of the body or cells may block the activity of the acetylcholine by hydrolizing it, thus regulating the effect of the acetylcholine. It also seems that an epinephrine-like substance is produced in the sympathetic nervous system and stimulates it at the junction of the sympathetic neuron and the cell (Cannon). The reaction of the active choline-like compound with the cell is ionic or saltlike; the cation replaces sodium, potassium or calcium ions of the cell. The combination of the cation and the cell changes the physicochemical activity of the cell. Atropine and pilocarpine offer different chemical structures. The antagonism between atropine and acetylcholine suggests different cell receptors or chemical antagonisms due to affinity between atropine and esterase of acetylcholine. An excess of potassium set free by cell activity might liberate acetylcholine. The ophthalmologist may be led into most interesting and important speculative fields by following the literature of this subject.[6]

The somewhat disassociated contraction of isolated dilator and sphincter fibers suggests a high summation. That is, to activate the muscle fibers a single nerve impulse would require an intermediate substance of the cholinergic or adrenergic type. The quick drop of response to atropine or acetylcholine by suspended muscle after changing the bath to fresh Ringer's solution indicates that the effect of these substances requires their continued presence about the muscle fibers and that they are not stored within the cells. Velhagen[7] has shown an active acetylcholine-like substance in aqueous, uvea and retina. He showed an active effect of this substance on the iris sphincter and an overactive atropine effect. The double innervation of sphincter has been suggested from several angles. Poos supported this idea from his valuable work on isolated iris and sphincter. Many authors have studied the effect of epinephrine on smooth muscles to the end of supporting the theory of double innervation. Joseph[8] demonstrated in living animals a relaxation of the sphincter on stimulation of the cervical portion of the sympathetic trunk. Our studies confirmed this from the in vitro angle.

We also demonstrated an indirect atropine effect on the dilator segment which was contracted by epinephrine. That is, the muscle plus

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6. Gaddum, J. H.: Gefässerweiternde Stoffe der Gewebe, Leipzig, Georg Thieme, 1936. Henderson, V. E., and Roepke, M. H.: Physiol. Rev. **17**:373, 1937. Brown, G. L.: ibid. **17**:485, 1937. Rosenblueth, A.: ibid. **17**:514, 1937. Eccles, J. C.: ibid. **17**:538, 1937. Alles, G. A.: Physiol. Rev. **14**:276, 1934. Loewi, O.: Arch. f. d. ges. Physiol. **193**:201, 1921; **204**:361, 1924. Poos, F.: Klin. Monatsbl. f. Augenh. (supp.) **78**:227, 1927.

7. Velhagen, K., Jr.: Arch. f. Augenh. **105**:573, 1932; **108**:126, 1933; **109**:195, 1935.

8. Joseph, D. R.: Am. J. Physiol. **55**:279, 1921.

epinephrine or neo-synephrin hydrochloride equals contraction (fig. 4). The muscle plus epinephrine, or neo-synephrin plus atropine equals *active* relaxation (figs. 5 and 6). The relaxed muscle plus atropine equals no change. Thus atropine either prevents the continual attachment of the sympatheticomimetic substance to the cell or its reaction from the attachment attained. This is assuming that the atropine has no effect on the dilator alone. It has been pointed out that in some vascular areas stimulation of the sympathetic system causes vasodilation due to the production of acetylcholine and that atropine removes

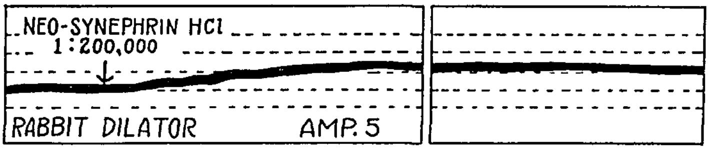

Fig. 4.—Rabbit dilator; neo-synephrin hydrochloride, 1:200,000; amplification, 5. The return to the base line may take as long as twenty minutes.

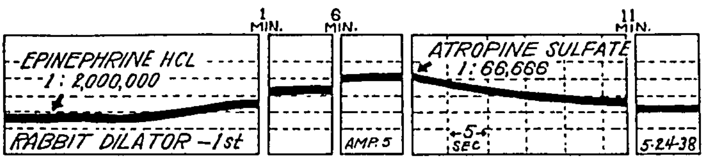

Fig. 5.—Rabbit dilator, fresh preparation; amplification, 5; epinephrine hydrochloride, 1:2,000,000; six minute interval; atropine sulfate, 1:66,666. A quick drop in the curve followed the administration of the atropine, indicating active antagonism.

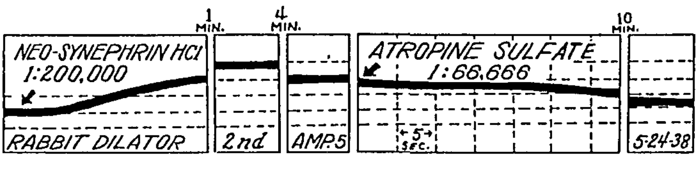

Fig. 6.—Rabbit dilator, second run (same preparation as in figure 5); amplification, 5; neo-synephrin hydrochloride, 1:200,000; four minute interval; atropine sulfate, 1:66,666. The atropine showed an active antagonism to the neo-synephrin hydrochloride.

this effect (Dale [9]). The depressing effect of atropine on the dilators of rabbits contracted by epinephrine, neo-synephrin or benzedrine was definite and antisynergistic. Preparations of isolated sphincter of the rabbit were examined for synergy. Subreactive dosages of homatropine hydrobromide, epinephrine, neo-synephrin or benzedrine were established. Then after contraction by acetylcholine these doses were introduced separately and successively and the effects noted on the curves at different points. A fresh sphincter will remain well con-

9. Dale, H. H.: J. Pharmacol. & Exper. Therap. **6**:147, 1914.

tracted and maintain a high rise in curve as long as fifteen minutes in a bath of acetylcholine in a dilution of 1:66,666. The *joint use* of homatropine and epinephrine or of homatropine and neo-synephrin, each in subeffective doses, applied at the beginnings of the peak of the acetylcholine response will depress such a fresh sphincter (fig. 7). But the response is not marked, and the curve maintains a gradual fall after a short initial drop. The length of time required for the curve to return to the base line is nearly equal to that after the administration of acetylcholine alone. We have more comparative studies in progress.

SOME CLINICAL SPECULATIONS

From the effect of heat on isolated iris muscle, its therapeutic value from prolonged use would be questionable. An active carrying in and out of blood and of antibodies in infection probably would be of value. But prolonged heat induces passive congestion and slows up local activity. If heat lowers the sphincter effect more than the dilator, some

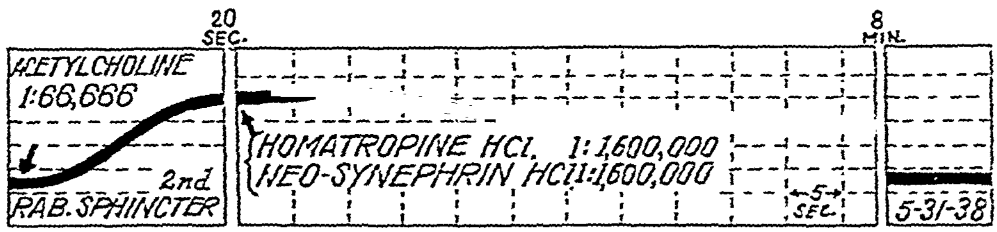

Fig. 7.—Rabbit sphincter, second run. A rise in the curve followed the use of acetylcholine, 1:66,666; hematropine hydrobromide plus neo-synephrin hydrochloride, each in a dilution of 1:1,600,000, caused a slight dip in the curve, followed by a slow return to the base line.

dilatation would follow its use. This would soon be offset by the larger area of congestion occupied by dilator sectors. Relief from the passive congestion could be partly and early had from cold. But the prolonged use of cold would again slow down the passage of fluid through the tissues. The letting of venous blood and the use of a strong decongestive drug, such as epinephrine or neo-synephrin, would be of more value. Blood in the anterior chamber in or behind the stroma of the iris may explain uneven and reluctant dilatation. When much blood is present in the anterior chamber and is of the dark, or tar, variety, a conservative measure is to evacuate it through incision, unless the opposite effect of immobilization is desired. The tearing of synechia by atropine is difficult to explain if this drug only relaxes the sphincter. The dilator fibers have substantial power for work, and it is possible that the formation of adhesions mechanically stimulates these fibers. A quicker, fuller dilatation and of shorter duration is had from a sympathetic dilator stimulus alone. Dilator stimulus plus atropine gives after a maximum response a drop back into a plateau. This drop is unlike the response from the sympathetic stimulator alone. One of the

most even ways to apply a drug, especially a sympathetic stimulator, is by an emulsion. Many of the variable effects obtained by some in clinical experimentation have been due to the winking out of the drops. Oil in water emulsion gives a fairly prolonged, even application to the cornea, through which the drug must be absorbed. Drugs that are strong sympathetic stimulators may be used in the field of ophthalmology, since the eyes are relatively resistant to them. To determine systemic reaction (rise in blood pressure) of an emulsion of 10 per cent neo-synephrin hydrochloride applied to the conjunctival sac or injected intravenously, an experiment on a dog was done. This showed little systemic effects with a 10 per cent emulsion (1 drop being equivalent to approximately 30 mg.) in the conjunctival sac as compared with the drastic effect of intravenous injection (fig. 8).[10]

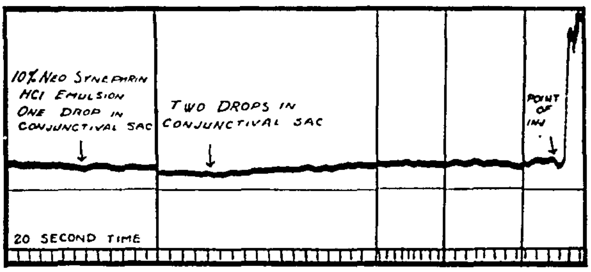

Fig. 8.—Comparative blood pressure effects in a dog to neo-synephrin hydrochloride administered into the cornea and intravenously. No significant rise followed a single application to the cornea, but a slight rise occurred after three doses. A single drop of a 10 per cent solution (about 30 mg.) administered intravenously caused a rise too high to record.

10. In this experiment a female dog weighing 6.5 Kg. was used. The anesthesia consisted of sodium barbital, 0.25 Gm. per kilogram of body weight; chlorobutanol, 0.1 Gm. per kilogram interperitoneally, and atropine sulfate, 0.05 mg. per kilogram hypodermically. The carotid artery was exposed and attached to a mercury manometer in the usual way, and recordings were made on a slowly revolving kymograph. The intravenous injection was made by way of the femoral vein. The results are shown in figure 8. 1. A normal blood pressure tracing is shown first. One drop of the emulsion (from the regular tube) was dropped on the upper part of the cornea, care being taken not to bring any pressure on the eyeball to avoid any vague stimulation. Little effect was noted. The sac was irrigated with Ringer's solution until the washings showed no trace of the emulsion. 2. After a fifteen minute rest, 2 drops of the emulsion was applied to the sac, causing a gradual rise within one minute. The maximum rise was approximately 6 mm. of mercury. This elevation was sustained after two ten minute rest periods (of the kymograph). The sac was then irrigated with Ringer's solution until the washings showed no trace of the emulsion. 3. After a forty minute rest, 1 drop of the emulsion dissolved in 0.6 cc. of Ringer's solution was injected into the femoral vein, causing an immediate rise of over 120 mm. of mercury (too high to record). The original blood pressure at the start of the experiment was 124 mm.

The sensitizing effect of one substance on another is of use in refraction, for which a quick, sharp response is wanted followed by a quick subsidence. The relative effect of neo-synephrin hydrochloride and benzedrine sulfate, each in a dilution of 1 : 50,000, on the acetylcholine-contracted sphincter of the rabbit may be seen in figures 9 and 10. The neo-synephrin produced considerably more depression of the sphincter. The effects of synergism of drugs may be due to double innervation; that is, with smooth muscles a stimulus may also cause a relaxation of an opposing muscle. Also, this effect may follow anatomic arrangement of the muscle fibers. The synergistic response to drugs may be due to the addible effect and also to sensitization, so that one

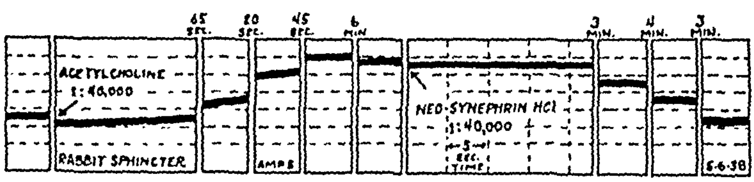

Fig. 9.—Rabbit sphincter; amplification, 5; acetylcholine, 1 : 40,000; neo-synephrin hydrochloride, 1 : 40,000. A rise in the curve occurred after the administration of the first drug and a fall to the base line after the administration of the latter.

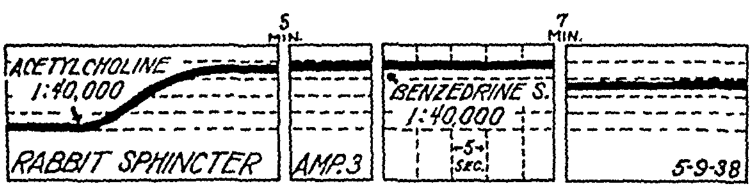

Fig. 10.—Rabbit sphincter; amplification, 3; acetylcholine hydrochloride, 1 : 40,000; benzedrine sulfate, 1 : 40,000. A rise in the curve occurred after the use of the acetylcholine, and the benzedrine sulfate failed to depress it to the base line.

drug increases tissue sensitivity to another. Removal of active inhibitors serves the same purpose. These possibilities are of practical value in therapeutics.

CONCLUSION

Isolated iris muscles, mostly of the rabbit, were investigated by a new autographic method. This method is extremely sensitive to physiologic and pharmacologic stimuli. An irregular spontaneous rhythm was found in the relaxed muscle. More rhythm was produced by a weakly loaded muscle. Mechanical stimuli produced contraction. The smooth iris muscles have qualities in general like those of other smooth muscles. The conclusions were reached that the sphincter and dilator do not oppose one another's action but cooperate. The impression was further gained that the total work power of the dilators was greater than that

of the sphincters. As far as our experiments went, the ion reactions as found by Poos were duplicated. A group of substances which stimulate the parasympathetic system, such as acetylcholine, mecholyl, physostigmine and pilocarpine, stimulated the sphincter but had no reaction on the relaxed dilator. A group of stimulators which affect the sympathetic nervous system, such as epinephrine, cocaine, ephedrine, neo-synephrin, benzedrine and paredrine (β-4-hydroxyphenylisopropylamine), were noted to stimulate the dilator fibers. Atropine and its group relaxed the sphincter and also relaxed the dilator when it was in a state of contraction due to epinephrine or neo-synephrin or benzedrine. A slight synergistic effect was found from sphincters contracted by acetylcholine, subminimal doses being used in combination, homatropine with either neo-synephrin or epinephrine or benzedrine. This was slight and rather variable. The total time depression effect on the contracted sphincter was questionable. An increase of tonus was accomplished by cooling, alkalization and mechanical stimulation and a decrease by acidification, heat, previous disease and age of tissue. The iris muscles could be sensitized by removal from the eye and kept in a cool state from four to thirty-six hours. No effects on the isolated iris muscles of the rabbit were found with insulin.

Miles Bruno, M.S., assisted with the experimental work.

CONGENITAL CYST OF THE VITREOUS

J. V. CASSADY, M.D.
SOUTH BEND, IND.

A review of the ophthalmic literature shows that congenital cyst of the vitreous is rare. Tansley [1] in 1899 described a cyst in the center of the vitreous chamber as follows:

> It would settle toward the disc or lens according to gravity following movement of the eye. It was irregularly spherical with small indentations, somewhat resembling a potato, and this simile was made still more positive by there being spots and lines of pigment upon the capsule. The spheroid was itself transparent or nearly so, and could only be seen by its pigment and capsule outline. The presence of the pigment upon its capsule would indicate that at some time there had been imflammatory action.

In a discussion of this cyst Dr. Alt offered the following explanation for its origin, which since then has been largely accepted:

> This is very interesting and in thinking of what might possibly give rise to such a cyst in the vitreous body, it occurs to me that perhaps a tumor that I have now found in eight eyes affecting one ciliary process only and looking like an adenoma undergoing mucoid degeneration and which I have described as adenoma of the ciliary process might give rise to such a cyst.

Dr. Randall offered at this time the explanation that cystic growths may be attached at the site of a coloboma. He considered this cyst one of "that character, formed at the point of a coloboma, loosened from its attachment and set adrift by a process of closure of the congenital cleft. It could be a vacuoled vitreous exudate."

Troncoso [2] in 1903 described a pigmented, transparent cyst of the vitreous which moved about in the vitreous, returning to the same place. His explanation was that it was a degenerated ciliary process, filled with aqueous humor, which had broken loose.

Shine [3] in 1913 presented a woman with a cyst of the vitreous before the ophthalmologic section of the New York Academy of Medicine. His description follows:

> The cyst was of steel gray color, floated freely about on movement of the eyeball, but on cessation of movement always seemed to return to the same point just behind the ciliary body, below or slightly to the temporal side.

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1. Tansley, J. O.: Tr. Am. Ophth. Soc. **9**:507, 1899.
2. Troncoso, U.: Ann. d'ocul. **30**:341, 1903.
3. Shine, F. W.: Arch. Ophth. **42**:398, 1913.

His explanation of its origin was that it was composed of epithelial tissue, an outgrowth of the ciliary body, detached or still attached to it by an invisible filament. Dr. Friedenberg had seen a similar case. The cyst was slightly pigmented and had a slow motion.

Scarlett[4] in 1929 described an oval translucent cyst one and one-half times the size of the disk in the anterior temporal quadrant of the vitreous. It consisted of two parts, joined by a constriction which gave the appearance of a cap. Sprinkled over the surface of the cyst were numerous black dots the size of a pinpoint. The cyst was freely movable in a fluid vitreous.

Hurwitz[5] in 1933 presented a girl with a cyst floating free in the vitreous before the Baltimore City Medical Society. The cyst was heavier than the vitreous, and when the patient held her face down the cyst would almost come up against the lens. It had many irregular spots of pigment on its surface. Hurwitz was fortunate in being able to see the cyst with the slit lamp. He stated:

> It appeared as a silver-white glistening sphere pigmented below with a shred or two of connective tissue running off loosely from its upper portion. My own feeling is that the cyst originated congenitally in a ciliary process and became detached into the vitreous.

Meding[6] in 1933 described a congenital cyst of the vitreous as follows:

> ". . . a round lens shaped body immediately posterior to the lens floating across the enlarged pupil and sinking slowly out of sight. Except for smudges of dark brown around its edges, the body was translucent and behaved very much like a dislocated lens. The slit lamp directed tangentially illumined the body beautifully, giving it the appearance of a gilded ball but afforded no penetration. Examined with the retinoscope the brown smudge proved to be granular deposits of pigment on the surface. The cyst moved up and down and remained in the line of vision more constantly in the morning. It was confined in its movement to a pathway or to a circumscribed shallow area. The circumscribed area in which it moved suggested that this body had existed from birth and was as transparent as the vitreous up to the date of its interference with vision; or it may have escaped or broken some attachments at that time.

He thought that if the cyst were of hyaloid origin there should have been some signs in the lens or elsewhere.

Seech[7] in 1933 presented a patient with a cyst of the vitreous before the Los Angeles Society of Ophthalmology and Otolaryngology. It

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4. Scarlett, H. W.: Tr. Am. Ophth. Soc. **27**:154, 1929.

5. Hurwitz, C. E.: Cyst in the Vitreous: Report of a Case, Arch. Ophth. **9**:825 (May) 1933.

6. Meding, C. B.: Free Cyst Floating in the Vitreous, Arch. Ophth. **11**:973 (June) 1934.

7. Seech, S. G.: Congenital Cyst in the Vitreous, Arch. Ophth. **11**:947 (June) 1934.

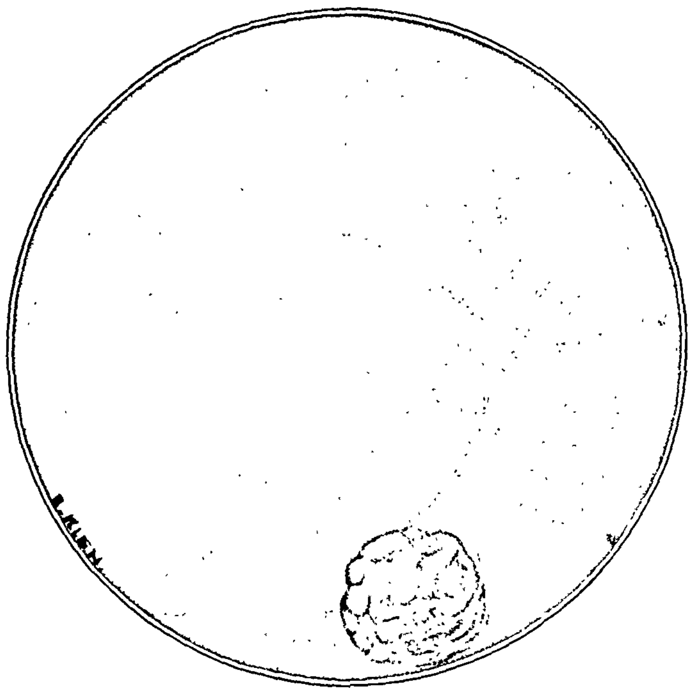

Fig. 1.—Congenital free floating cyst in the vitreous. The fundus is seen indistinctly, as the cyst is in focus with the ophthalmoscope. The shiny hyaline appearance is best seen over the edge of the pupil, below. Similar lines appear dark above as a silhouette against the background. The prepapillary membrane is at the lower nasal side of the disk.

was a slightly oval cyst in the lower half of the vitreous, divided into two parts by a constriction. According to Seech:

> The cyst showed considerable black pigment in several places apparently on its inner surface. There were numerous dots of pigment scattered around in this part of the vitreous and the fibrils of the anterior part of the vitreous were absent.

McLean,[8] Brewerton,[9] Koller,[10] Hill,[11] Lacarrere,[12] Litinsky,[13] Perara[14] and Vennin[15] described cysts of the vitreous occurring in eyes with retinitis pigmentosa or other degenerative or inflammatory diseases of the fundus, and although they may have been congenital, they were not of necessity such. Frequently degenerative or inflammatory changes produce such a cyst. Litinsky, McLean, Vennin and Perara, however, in my opinion gave a better explanation of the origin of these congenital cysts than most observers. They were of the opinion that the cysts originated from the fetal ocular cleft or the mesodermal elements of the embryonic vitreous or hyaloid apparatus.

Other observers have reported cysts attached at or near the optic papilla but not floating free in the vitreous. Such cysts probably are more closely related to the cysts floating free in the vitreous than are cystic degenerations of the ciliary processes, which have been so often accepted as the site of congenital free floating cysts in the vitreous.

Koller[10] in 1901 described a cyst of the vitreous as follows:

> The lower one-third of the disc appears to be hidden by a dazzling-white, egg-shaped body. Extending from this body were two short and one long process. The long process extended forward as a hyaloid artery and ended blindly near the nasal lower quadrant of the lens. There was a history of trauma in childhood.

He explained this cyst as evidently a widening of the hyaloid artery and its sheath at its posterior extremity, where it emerges from the optic nerve.

Schwartz[16] in 1936 presented a patient with a cyst of the vitreous attached to the retina before the Clinical Society of the Harlem Eye and Ear Hospital. Directly below the papilla there was a more or less

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8. McLean, A. L.: Am. J. Ophth. **16**:51, 1933.

9. Brewerton, E. W.: Tr. Ophth. Soc. U. Kingdom **33**:93. 1913.

10. Koller, C.: Tr. Am. Ophth. Soc. **9**:380, 1901.

11. Hill, E., in discussion on Scarlett.[4]

12. Lacarrere, J. L.: Nota sobre un caso de formaciones quisticas hialoides, inflammatorias? simetricas prepapilares, in Concilium ophthalmologicum, 's Gravenhage (The Hague), Netherlands, 1929.

13. Litinsky, G. A.: Klin. Monatsbl. f. Augenh. **87**:205, 1931.

14. Perara, C. A.: Bilateral Cyst of the Vitreous, Arch. Ophth. **16**:1015 (Dec.) 1936.

15. Vennin: Lyon méd. **119**:974, 1910.

16. Schwartz, L. H.: Cyst of the Vitreous Attached to the Retina. Arch. Ophth. **16**:230 (Aug.) 1936.

transparent bluish white swelling about two times the size of the disk. The cyst was adherent to the retina and to the central retinal vessels. The surface of the cyst was slightly pitted.

Derby[17] described a pear-shaped translucent cyst, slightly smaller than the disk, with a greenish reflex, in a man aged 40. The cyst consisted of a rounded prominence on the temporal side of the disk.

Stieren[18] in 1933 reported a cyst on the nerve head of a 6 year old Negro girl. About 2 disk diameters above the macula there was a bluish white cyst about 1½ disk diameters in circumference which seemed to arise in opaque nerve fibers. It was elevated about 5 diopters and resembled a frosted light bulb.

REPORT OF A CASE

The cyst that I observed was discovered accidentally. A report of this case follows:

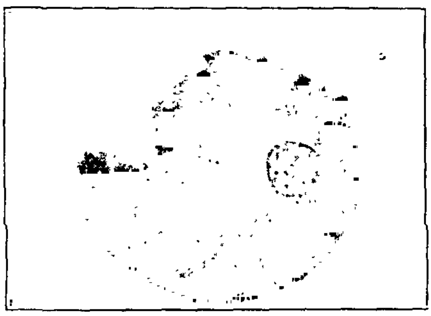

Fig. 2.—The cyst in focus. The vessels of the fundus can be seen through the transparent cyst.

A young married woman of 25 came to me in February 1938 because of headache. During an indirect examination of her fundus with an undilated pupil a shadow was seen floating across the fundus. She had never had ocular symptoms, and the cyst had never interfered with vision. The vision was 20/20, and under cycloplegia with homatropine hydrobromide, she accepted only a + 0.50 sphere in each eye, with vision of 20/20.

Examination of the right eye showed a prepapillary membrane extending over the nasal edge of the disk like a veil, nearly hiding the vessels, but they could be seen indistinctly through it and appeared normal. The remainder of the fundus was normal with the exception of a round floating shadow, like that of a cloud on the ground of a sun-lighted field. The shadow was cast by a round semitransparent cyst floating freely in the vitreous, and it was seen best with a lens of from 8 to 10 diopters in the ophthalmoscope. Observed directly, it seemed to have smudges of pigment over its edges and streaks and spots of pigment over its surface. If observed more closely over the edge of the dilated pupil, with some of the light reflected from the iris striking it, it appeared as a gilded, shiny pearl-like ball. It seemed to have plaques of hyalin-like deposit in its transparent capsule. It rotated on its vertical axis, was freely movable and was slightly

17. Derby, G. S.: Tr. Am. Ophth. Soc. **12**:827, 1909-1911.
18. Stieren, E.: Tr. Am. Ophth. Soc. **31**:116, 1933.

heavier than the vitreous, sinking downward out of sight when the eye was quiet. When the patient put her head forward or backward the cyst could not be made to approach either the lens or the fundus but seemed limited in its motility to the central and lower portions of the vitreous. The cyst was transparent. The vessels of the fundus as well as the posterior capsular wall could be seen through it. The capsule of the cyst was thin and was seen only by contrast with the surrounding vitreous.

The remainder of the vitreous was normal; the other media, the lens, aqueous and cornea, were clear. The slit lamp did not reveal the cyst or show any abnormalities in the vitreous.

Examination of the left eye revealed a slight prepapillary membrane, but not as marked as that of the right eye. The remainder of the fundus and the media were normal.

Since then I have observed this cyst at frequent intervals. There have been no changes in the appearance of the fundus or the cyst or in the visual acuity of the affected eye. Because of this observation, I feel justified in presenting this case as one of congenital free floating cyst of the vitreous.

COMMENT

The apparent pigmentation of the cyst is an illusion. Observed on the light background of the fundus, the shiny hyalin-like plaques of its surface appear in silhouette as streaks and spots of dark pigment. Observed directly, with the light reflected from the edge of the pupil, as is best seen in the drawing, this may be wrinkling of the capsule of the cyst, as it has contracted in size. Shine [3] reported the cyst that he observed as of steel gray color. Hurwitz,[5] in observing his reported cyst with the slit lamp, described it as a silver-white glistening sphere. Meding,[6] who observed his cyst with the slit lamp, described it as having the appearance of a gilded ball.

In reviewing the origin and development of the vitreous, it seems probable that the cysts are remnants of the primary vitreous rather than detached ciliary processes.

Their usual location in the central and lower part of the vitreous corresponds to that of the hyaloid remnants. The ciliary body and the tertiary vitreous are separated from the main body of the vitreous by condensation and Egger's line. This would almost preclude the possibility of such a cyst located in the central portion of the vitreous being a cyst of the ciliary process. The primary vitreous has the degenerated hyaloid remnants with its glial sheath from the ectoderm of the optic disk and its mesodermal elements. This seems most likely to be the origin of the cyst described here as well as of other previously described cysts of the vitreous.

The pigmented appearance of the cyst is probably formed by its silhouette on a light background. Examined directly at the edge of the pupil rather than through the center, with light reflected from the margin of the iris, the cyst appears shiny, glistening and unpigmented.

An apparent wrinkling of its surface gives it areas of greater wall thickness, like hyaline plaques. These areas, viewed through the center of the pupil with the cyst appearing in contrast with the light background and illuminated by reflected light from the fundus, appear dark. This was borne out in Shine's case in which he was able to observe the cyst with the slit lamp (a globular, semitransparent, grayish cyst floating freely in the vitreous).

On the basis of the newer knowledge of the origin and development of the vitreous into primary, secondary and tertiary vitreous, described by Mann and others, the separation of the ciliary body from the usual site of such a cyst and the absence of pigment, the interpretation of the origin of these cysts as being necessarily "a degenerated adenoma of the ciliary process which broke loose into the vitreous," as considered by Alt, should be revised. Troncoso also was of the opinion that cysts in the vitreous originate from the uvea, which accounted for the accumulation of pigment on the surface of the cyst.

The associated prepapillary membrane in the case reported here suggests a remnant of the primitive epithelial papilla of Bergmeister, which forms the sheath of the hyaloid arteries and its branches. The presence of this membrane, the lack of pigment and the location of the cyst in the center of the vitreous suggest the primary vitreous as the site and origin of the cyst as opposed to the theory previously advanced, that the cyst probably originated from a degenerative adenomatous cyst of the vitreous processes.

OCULAR MANIFESTATIONS IN BRUCELLOSIS (UNDULANT FEVER)

JOHN GREEN, M.D.

ST. LOUIS

The following brief historical account of Malta fever (undulant fever, brucellosis) is given in a paper by Baltzan:[1]

An epidemic form of fever in man had long existed in the Mediterranean area and was especially noticed in the island of Malta. It was first described by Marston in 1861 and called "Mediterranean fever." But it was not until 1886 that Sir David Bruce proved the etiological factor, which he called *Micrococcus melitensis.* In honor of Bruce other organisms belonging to this group have been given the generic name of Brucella. Twenty years later it was recognized that the goat was the host, and that in herds this organism was the cause of abortion. In the human being the disease was variously termed Mediterranean, Gibraltar, Malta, undulating, and undulant fever. It was reported in America as early as 1897 and since epidemics were restricted to regions where goats were largely handled, it received the popular name of "goat fever."

Bang, a Danish veterinarian, succeeded in isolating the causative agent of abortion in cattle in 1897. Its pathogenicity for man was, however, not fully recognized until nearly twenty-five years later, although earlier, in 1911, Schroeder and Cotton reported finding the abortion bacilli of Bang in milk sold for consumption, and suggested this might result in human infection. In 1914 Traum isolated the causative agent of abortion in swine. The question was not raised for some time afterwards whether there was any relationship between these organisms either to one another or to man.

Heretofore, three organisms were known to affect separately abortion in goats, cattle, and dogs. Only one of these, *Brucella melitensis,* the caprine or goat variety, was already proved a cause of fever in man. In 1918, Alice Evans, at Washington, demonstrated the intimate morphological, cultural, and biochemical relationships of the goat and cattle (Bang's) organisms. In consequence of this investigation the Brucella-like organisms originating from goats, cattle, and swine were designated as varieties of the same genus. Then, in 1924, Keefer in Baltimore reported a case of undulant fever in a patient who had had no contact with goats. The infection was the bovine type. Since then the porcine strain has also been recovered in human disease, and even known to be more virulent and more readily acquired from contaminated material, and not only by ingestion.

A perusal of Bruce's[2] brief paper, "Notes on the Discovery of a Microorganism in Malta Fever," compels admiration for this British

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Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 9, 1938.

1. Baltzan, D. M.: Experience with Fifty-Seven Brucellosis Infections in Saskatchewan, Canad. M. A. J. **36**:258, 1937.

2. Bruce, D.: Note on the Discovery of a Microorganism in Malta Fever, Practitioner **39**:161, 1887.

army surgeon who, practically unaided and with primitive laboratory equipment, gave impetus to a train of investigation which has led to results of immense importance to veterinary and medical science.

In 1918 Alice Evans [3] established the essential identity of Brucella melitensis and Brucella abortus. The following statements from her paper are, in the light of the later discovery of the pathogenicity of Br. abortus for human beings, truly prophetic:

> The very close relationship between B. abortus and an organism pathogenic to human beings adds new interest to the subject of the possible pathogenicity of B. abortus to human subjects. Considering the close relationship between the two organisms and the reported frequency of virulent strains of B. abortus in cow's milk, it would seem remarkable that we do not have a disease resembling Malta fever prevalent in this country. . . . Are we sure that cases of glandular disease, or cases of abortion, or possibly diseases of the respiratory tract may not sometimes occur among human subjects in this country as a result of drinking raw cow's milk?

Up to 1924 interest in the disease was confined largely to veterinarians. After Bang's discovery of Br. abortus, investigations were carried on in veterinary laboratories throughout the world to determine the incidence of the disease in herds of cattle. It is conservatively estimated that between 15 and 20 per cent of cattle in the United States are infected with this organism. Many seemingly normal animals are carriers. If an animal aborts it does not ordinarily show other clinical signs of the disease.

Carpenter and King [4] found that 20.4 per cent of the raw milk supply of three counties in central New York showed infection with Br. abortus. Gilbert and Coleman [5] studied undulant fever in New York state, where two million people have a raw milk supply. Several hundred cases of undulant fever are diagnosed annually. They expressed the belief that only one tenth of the actually existing cases are recognized. In most of the cases in New York the disease was due to organisms of the bovine type. Most of the patients had no contact with cattle. Consequently, raw milk seemed to be the source of the trouble.

Keefer [6] was the first to isolate Br. abortus from the blood of a human being who had suffered from a continuous fever of several weeks' duration.

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3. Evans, A.: Further Studies on Bacterium Abortus and Related Bacteria: II. A Comparison of Bacterium Abortus with Bacterium Bronchiseptus and with the Organism Which Causes Malta Fever, J. Infect. Dis. **22**:580, 1918.

4. Carpenter, C. M., and King, M. J.: Symposium on Undulant Fever, New York, American Public Health Association, 1929.

5. Gilbert, K., and Coleman, M. B.: Recent Cases of Undulant Fever in New York State, J. Infect. Dis. **43**:273, 1928.

6. Keefer, C. S.: Malta Fever Originating in Baltimore, Bull. Johns Hopkins Hosp. **35**:6, 1924.

Human beings may become infected (*a*) by consuming unpasteurized milk or other dairy products containing Br. abortus and (*b*) by infection through abrasions in the skin and other wounds. From the digestive tract the organism invades the mucous membrane, passing thence into the blood stream or indirectly by way of the lymphatic system. The lymphatics and the blood stream may become invaded by infection from the tonsils. Carpenter and Boak [7] found the organism in 8 of 56 pairs of human tonsils after surgical removal. This finding has been confirmed by other investigators.

Great differences in susceptibility exist. For instance, only one infection may occur in a family although all the members may be drinking the same milk. Men working in abattoirs may develop an active immunity; others, working under the same circumstances, may contract the disease. These infections could almost certainly be prevented by the workers' wearing rubber gloves. From 5 to 7 per cent of persons living in the country or in smaller towns where most of the milk sold is not pasteurized carry Br. abortus antibodies in their blood. Some of these have developed an active immunity resulting from an unrecognized mild infection not causing symptoms of the disease.

The Br. abortus is found in goats, sheep, swine and horses. The bovine strains are extremely pathogenic for the human family. Attempts have been made in Europe to immunize laboratory workers by injecting intracutaneously 0.1 cc. of a suspension containing approximately 5,000,000 killed organisms per cubic centimeter. There is good evidence to show that 50 per cent of all veterinarians are infected.

Hughes [8] in 1897 described three general types of this infection: (1) the persistent type in which the fever continues intermittently for several weeks or months; (2) the undulant type, marked by exacerbations of fever at regular intervals; and (3) the pernicious or fulminating type in which the temperature rapidly rises to 106 F. or even higher. The last type, rare in this country, is, as a rule, fatal. Evidence is accumulating to show that the first and second types may drift into a chronic form, enduring for many years. The sufferer may have long periods of well-being, followed by periods of lassitude, malaise, generalized weakness, arthritic pains and headache.

The period of incubation is from five to fifteen days. The onset is gradual, with weakness, general malaise, headache, backache, anorexia and constipation, followed by fever, chills, sweats, aching of the muscles, bones and joints and loss of weight. One characteristic is that the

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7. Carpenter, C. M., and Boak, R. A.: The Isolation of Brucella Abortus from Tonsils, J. A. M. A. **99**:296 (July 23) 1932.

8. Hughes, M. L., cited by Bierring, W. L.: Undulant Fever: Clinical Characteristics Based on a Study of One Hundred and Fifty Cases Observed in Iowa, J. A. M. A. **93**:897 (Sept. 21) 1929.

appearance and general condition of the patient may be surprisingly good despite a rapid pulse and high temperature.

Physical signs are enlargement of the spleen and lymph glands (less frequent), swelling of the joints without redness and pain and abdominal pains suggestive of appendicitis or cholecystitis.

Harris [9] expressed the belief that "clinically Brucellosis is to be suspected in any obscure acute or chronic ailment. The symptomatology is tremendously variable and in any given case may simulate any one of a long list of acute and semi-acute diseases." This author listed 41 diseases with which it may be confused. He stated further: "The infection may be so mild as to completely escape detection and indeed may exist in thousands of patients in so minimal a degree that the patient never seeks medical advice. . . . These mild infections may at any time become virulent after months or years of latency."

Two of my internist colleagues in St. Louis have been applying a cutaneous test for brucellosis to patients who are seen now and again with all sorts of vague and unexplainable symptoms—the type of patient who makes life miserable for the physician. In a surprisingly large number of such patients a positive reaction has been elicited to this test for present or past infection with Br. abortus.

DIAGNOSIS

In the present state of knowledge a positive diagnosis of brucellosis cannot be made on clinical appearances alone. The aid of a good pathologic laboratory manned by pathologists conversant with the technic of the various tests is essential.

Opsonocytophagic Test.—In order to inhibit the normal opsonins, the blood of the patient is mixed with sodium citrate. Two-tenths cubic centimeter of 20 per cent sodium citrate in saline solution is prepared. To this is added 5 cc. of blood. A standard strain of Brucella which has been incubated for forty-eight hours is mixed with the citrated blood, and the mixture is incubated at 37 C. for one hour. Ordinary blood smears are then made and stained. Fifty white cells are examined to determine the number of organisms that have been picked up by each cell. From this is calculated the Foshay [10] opsonocytophagic index. In most cases the index number seems to parallel roughly the state of the patient's resistance to the disease.

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9. Harris, H. J.: Undulant Fever (Brucellosis): Difficulties in Diagnosis and Treatment; Supplementary Report on Fifty-One Cases with Observations on One Hundred and Twenty Additional Cases, New York State J. Med. **37:**1295, 1937.

10. Foshay, L., and Le Blanc, T. J.: Derivation of Index Number for Opsonocytophagic Test, J. Lab. & Clin. Med. **22:**1297, 1937.

Blood Agglutination Test.—The agglutination test of the blood is of value only if it is positive. In the vast majority of cases of chronic brucellosis it is negative (over 90 per cent in one series). Ordinarily an agglutination titer is found relatively early and only during the acute stage of the disease. Agglutination tests may remain negative during the entire course of the disease. As Harris [9] stated: "The partial agglutination with B. abortus in patients who present a clinical picture of Brucellosis is just as significant as is agglutination in higher dilutions of the serum. To ignore this point is to overlook a majority of the cases of Brucellosis that come under observation." It should be remembered that the agglutination test for brucellosis is not a test for the presence of organisms but only for the determination of specific agglutinins in the blood.

Cutaneous Test.—A small quantity of killed bacillary suspension or Huddleston's brucellin is injected intradermally into the forearm. The reaction begins within twenty-four hours or even earlier and appears as a rapidly spreading area of redness with an indurated center, usually with considerable edema. Heat, pain and itching are present as a rule; infrequently, sloughing of the superficial tissues occurs. Any reaction persisting to the third day is considered positive.

After the cutaneous test the patient's reaction is variable. Twenty-five per cent show no reaction; 10 per cent feel better. In 65 per cent there is an intensification of all symptoms. Rarely severe constitutional reactions follow.

A positive reaction to any laboratory test does not prove the existence of active brucellosis but indicates that the patient has or has had the disease. The cutaneous test is the most sensitive of the tests and is of great value in detecting chronic inactive brucellosis with few or ill defined symptoms.

THERAPY

The therapy for brucellosis has not kept pace with the rapid increase in the knowledge of the symptomatology, bacteriology and laboratory diagnosis of the disease. Chemotherapy has proved disappointing. Fever therapy, by means of the Kettering hypertherm, has been effective in some cases of acute brucellosis. It probably acts by raising the phagocytic power of the blood. According to Newman: [11] "Possibly certain chemical changes take place within the tissues which either stimulate immune reactions or else render the tissues unsuitable for the continued growth and reproduction of the bacilli."

Specific serums and commercial vaccines have not always proved effective. Recently Dr. Lee Foshay, of the Cincinnati General Hos-

11. Newman, H. G.: Diagnosis and Treatment of Undulant Fever, Radiol. Rev. & Mississippi Valley M. J. 60:31, 1938.

pital, developed an effective serum for the treatment of the disease in the acute stage and a vaccine for treatment in the chronic stage. The serum and vaccine are not yet on the market and hence not available to the general medical profession.

The occurrence of ocular manifestations in the course of brucellosis is now well established. It is my impression that comparatively few ophthalmologists are taking advantage of the opportunity to determine by laboratory methods the presence or absence of past or present infection with Brucella. Much information can be gained by applying the simple intradermal test now available. Full data, however, can be secured only with the aid of a laboratory equipped to perform the opsonophagocytic and agglutination tests.

REPORTS OF CASES

CASE 1.—In 1917 H. G. N. was employed as a research assistant in animal pathology at the Missouri State Agricultural College. His work consisted of handling laboratory cultures of abortion bacilli from both cattle and swine. He was constantly exposed to infected experimental animals both in the field and in the laboratory. At that time the bacillus was considered harmless to man; consequently no precautions were taken to prevent infection. While thus engaged, he became ill with a strange malady which began with fever, weakness and night sweats. His right eye became inflamed; the diagnosis was phyctenular conjunctivitis. Tuberculosis was suspected but ruled out by roentgenographic and routine examinations.

After these symptoms had persisted for several months, there developed a high fever and a marked cutaneous rash which was regarded as scarlet fever. The absence of lesions in the throat and the disappearance of the rash in a few hours led to the abandonment of this diagnosis. Next appeared marked jaundice and clay-colored stools, finally terminating in the patient's recovery after one day in coma.

In 1918 the man had severe tonsillitis, followed by influenza with pneumonia. After recovery he was restored to comparative good health. Repeated attacks of sinusitis added to his discomfort. In 1925 his tonsils were removed, and later, because of tenderness in the right lower abdominal region, appendicitis was suspected and an appendectomy was performed.

He first came under my observation in April 1930, when he was a medical student in St. Louis. At this time he presented in the right eye a small phlyctenule adjacent to, but not invading, the cornea, at 4 o'clock. Four days later the eye was well. In March 1931 he returned with a faintly staining marginal corneal lesion extending from 3 to 4 o'clock. Again recovery occurred in a few days. A similar but even less marked attack occurred on Nov. 12, 1934; and again there was a slight attack in April 1935. He felt that there was some connection between the exacerbations of sinusitis and the corneal manifestations; during each attack the rhinologist shrunk his nasal mucosa and on one occasion washed out his right antrum. In January and February 1936 he had his most severe attack of corneal trouble. A linear marginal corneal ulcer developed at 4 o'clock. The ulcer continued to spread upward and downward, and five days later about one sixth of the circumference of the cornea was involved. He was hospitalized and given general ultraviolet therapy, and the lesion was exposed to ultraviolet

radiation with the Hildreth lamp. (Dr. H. Rommel Hildreth carried out this part of the treatment.) The eye was kept atropinized for two and a half weeks. The lesion finally healed completely. There have been no subsequent recurrences.

In the summer of 1937, twenty years after the acute episode, the man volunteered to act as a control for a cutaneous test for undulant fever made on a subject who was thought to have this condition. This person had no reaction, but the control had, within twenty-four hours, a general reaction, with chills, fever and severe pain in the muscles for a whole day. In addition, a marked local reaction occurred, with swelling, induration and edema around the point of injection, which finally ended in a deep slough. An opsonocytophagic index showed that over 90 per cent of his leukocytes were picking up abortus bacilli. A low white cell count with a high percentage of monocytes was also present, this blood picture being typical of the disease in the chronic stage. The symptoms subsequent to the period of acute infection consisted of recurring attacks of pain in the muscles, joints and nerves. These attacks occurred on an average of three or four weeks. Great improvement occurred after the administration of Foshay's vaccine.

Case 2.—Mrs. J. F. R., aged 51, came under observation on Jan. 10, 1938, with the complaint of disturbed vision in the right eye. Vision in this eye was 6/7.5 with a + 2.25 sph. There was definite metamorphopsia. Ophthalmoscopic examination showed the retinal veins to be engorged but not tortuous. The arterioles in the macular region were numerous and tortuous. In the lower part of the macular region and encroaching on the fovea there was a slight subretinal elevation, about ⅔ disk diameter. The upper part of the lesion skirted the fovea, and here there were a few small round hemorrhages. The left eyeground was normal, and vision with correction was normal. Her physician reported that her general condition as excellent.

At this time she was aware of the existence of a number of bad teeth, and during February and March three upper and five lower teeth were extracted. Vision dropped to 6/30, eccentric. The reactions to the Mantoux test with two dilutions of tuberculin were entirely negative, as were the reactions to the Wassermann and Kahn tests. The opsonic tests with Br. abortus showed a Foshay index of 23, indicating a low resistance. The reaction to the cutaneous test for Br. abortus was strongly positive. So far there has been no opportunity to try out the specific vaccine on this patient.

Case 3.[12]—K. W., a housewife aged 70, entered St. Luke's Hospital early in 1937. Three or four weeks prior to entrance she observed a swelling about the size of a pea on the right side of her forehead just within the hair line. This extended in the direction of her right ear and was tender to pressure and painful at night. There was a constant fever, the temperature ranging from 100.5 to 101 F.

Examination showed firm cordlike structures on both sides of the forehead running up to the midline of the frontal region. They were not connected. They were probably situated within the skin, but their true nature was not determined. Shortly after the patient's admission to the hospital, Dr. Shahan found acute optic neuritis in the right eye, with partial loss of the left field. Vision was reduced to 8/30. Three abscessed teeth were extracted. Neurologic examination gave essentially negative results. There were no nasal appearances to warrant treatment or operation. Within a few days the optic neuritis showed improvement, and the patient was dismissed from the hospital.

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12. Dr. William E. Shahan gave me permission to report this case.

On reentrance five weeks later she complained of pain in many joints and in the muscles of the thighs. There had been considerable pain on motion, but at the time of her admission this had ceased. There were also night sweats and an afternoon rise of temperature and headaches. A vaccine made from cultures of Br. abortus was injected intradermally. A marked induration and a large area of erythema adjacent to the site of injection followed. Dr. Shahan found that the optic neuritis had subsided, leaving a slightly pale disk. The patient was put on small doses of Foshay's Br. abortus vaccine, which were continued by her physician for several months, with marked improvement in the symptoms. The Foshay index rose from 61 to 87 in three months.

The agglutination tests were negative for Bacillus typhosus, Bacillus paratyphosus A and B, Br. abortus and Br. melitensis. The reactions to the Wassermann and Kline tests were negative. A roentgenogram of the chest showed evidence of interlobar pleurisy and arteriosclerotic arcus aortae.

Case 4.[13]—A married woman aged 34 complained of a feeling of malaise. She suffered from general loss of energy and a sense of discomfort from the late spring of 1934 till August of the same year. There was no examination or treatment until August, at which time she noticed a blurring of vision in the left eye. At this time she was found to have a daily low grade fever, the temperature ranging from 99 to 100 F. The vision in the left eye was 20/70, owing to a central retinochoroiditis. Examinations in late August 1934 were positive for undulant fever. During September and October she received antimelitensis vaccine (Simpson). This had little appreciable effect on her general symptoms or on the ocular condition. In January 1935 she received 20 cc. of antimelitensis serum (Foshay) in two doses intravenously. There was a questionable abatement of symptoms for the next two or three weeks. She received no more treatment, but in the course of several months the fever and general debility subsided, until by the fall of 1935 she was free from all systemic symptoms. The condition of the eye remained stationary from August 1934 to October and then gradually improved. By the late spring of 1935 the vision had improved to 20/25 and has remained so.

I have been able to gather from the literature the following instances of ocular complications appearing in cases of undulant fever. In each case the clinical diagnosis was confirmed by one or more of the laboratory tests and also by the exclusion of other infections that might conceivably have borne a causative relation to the ocular malady.

Aurand's Case.[14]—The patient, a woman, sought consultation for diminution of vision. She had been ill for ten months. The original diagnosis was "grip." Various treatments failed to check her malady, which was characterized by continued fever, great thirst and anorexia. Culture of the blood revealed melitococci. In the course of the disease, which was finally treated with antimelitococcic vaccine, iritis developed, which finally yielded, leaving deposits of fine pigment on the anterior capsule of the lens. The visual acuity was little impaired.

13. Dr. H. G. Newman gave me permission to report this case, which was observed by Dr. S. C. Fulmer, of Little Rock, Ark.

14. Aurand, L.: Iritis au cours d'une fièvre de Malte, Bull. Soc. d'opht. de Paris, April 1936, p. 393.

Aubaret and Roger's Case.[15]—A woman aged 36 had fever of three months' duration accompanied by profuse perspiration, headache, stiffness of the neck, nausea, epigastric pain, constipation and backache. Early in the disease the vision deteriorated. In eight or ten days blindness was almost complete but was followed by slight improvement. Laboratory examination eliminated malaria, typhoid and syphilis; melitococci were found on culture. Vision was 1/50 in each eye. Ophthalmoscopic examination showed atrophy of the optic nerve, with narrowed vessels. There were signs of meningeal irritation. A variety of treatments failed to help. Five months after the onset of the disease melitococcic vaccine was used, followed by improvement in the general condition and slight improvement in the vision.

Villard and Pech's Cases.[16]—Case 1.—A girl of 15 had a moderate fever. Culture of the blood revealed melitococci. Three weeks after the beginning of the illness the right eye became red, and vision rapidly diminished. Hypopyon was noted. There were numerous synechiae, which yielded to atropine. A yellowish mass was found in the vitreous. In spite of vigorous treatment with foreign proteins, intravenous injections of mercuric cyanide, etc., sight was lost, and the eye eventually atrophied.

Case 2.—A woman aged 33 had loss of weight accompanied by cough. The tentative diagnosis was tuberculosis. Culture of the blood showed melitococci. Three months later the right eye became painful. Examination showed an acute iridochoroiditis with a blocked pupil. Vision was limited to perception of light. Treatment was unavailing, and blindness was complete five months later.

Bergmark's Case.[17]—A man aged 55 began in May 1938 to have severe headache and a continuous fever. The liver and spleen were palpable. Agglutination and complement fixation tests, both with the blood and with the spinal fluid, were positive for Br. abortus. In the right fundus oculi there was a small hemorrhage and beside it a greenish yellow exudate. The diagnosis was septic retinitis. There were many neurologic signs. Eight months after the patient's admission to the hospital the optic nerve on both sides was well advanced toward atrophy. Death occurred from intestinal hemorrhage two years after admission. Autopsy revealed chronic meningoencephalitis.

Bingel and Jacobsthal's Case.[18]—A man aged 32, who had been drinking raw milk frequently for several months, complained of general fatigue, lack of interest in life, loss of appetite and occasional headache. A severe headache suddenly developed, accompanied by vomiting and fever, the temperature being high. On examination of the nose and sinuses nothing was found. Examination of the eyegrounds showed choked disks amounting to 2 or 3 diopters. Culture of the blood revealed Br. abortus. Later cultures of the spinal fluid also revealed the organism. The history is incomplete in regard to the outcome of the ocular condition.

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15. Aubaret, E., and Roger, H.: Névrite optique, mélitococcique, Rev. d'oto-neuro-opht. **9**:50, 1931.

16. Villard, H., and Pech: Deux cas d'ophthalmie métastatique d'origine mélitococcique, Bull. Soc. d'opht. de Paris, December 1929, p. 676.

17. Bergmark, G.: Ueber Nervensymptome bei Febris undulans (Bang), Acta psychiat. et neurol. **6**:387, 1931.

18. Bingel, A., and Jacobsthal, E.: Ueber Meningitis bei Banginfektion: Ihr klinisches und bakteriologischserologisches Bild, Klin. Wchnschr. **12**:1093, 1933.

CANNAVÒ'S CASE.[19]—The patient aged 26 contracted the disease while in contact with herds of sheep and cows, supposedly infected with Brucella. His trouble began in July 1934, with continued fever and pain in the joints. All serologic tests other than for Brucella gave negative results. The serologic test for Brucella was positive with a dilution of 1:200. Between the fortieth and the fiftieth day of the illness vision in the left eye deteriorated. A little later a diagnosis was made of choroiditis with floating material in the vitreous. Cannavò is positive that all etiologic factors other than Brucella could be ruled out. Vision was finally reduced almost to blindness.

COHEN BOULAKIA'S CASES.[20]—CASE 1.—A youth aged 18 had undulant fever of eight months' duration. The diagnosis was confirmed by positive laboratory tests. For fifteen days he had noticed a veil in front of his left eye. This phenomenon lasted two or three hours and then disappeared. Vision decreased rapidly to counting of fingers at 30 cm. Ophthalmoscopic examination showed that the edges of the disk were edematous. Eight days later the entire retina was diffusely edematous. Still later there were vast plaques of retinal hemorrhage. Finally, massive hemorrhages into the vitreous occurred, with secondary glaucoma, necessitating iridectomy. The final result was atrophy of the optic nerve.

CASE 2.—A youth aged 17 during recovery from melitensis complained of diminution of sight in the right eye. Ophthalmoscopic examination showed a grayish discoloration of the macular zone. This was surrounded by a small whitish halo, equal on the two sides and assumed to be of congenital origin. The amblyopia lasted three weeks, and vision was completely restored.

GODWIN'S CASE.[21]—A woman aged 55 had been ill two and a half months and had had a temperature of about 103 F. for the first three weeks of that time. The diagnosis of Malta fever was confirmed by agglutination tests. Vision in the right eye was 6/20, eccentric. She presented a definite papillitis in the right eye—edema of the nerve head and tortuosity of the retinal veins. The left fundus showed slight blurring of the margins of the disk and moderate tortuosity of the veins. After initial improvement, there was a relapse. Full vision was not regained.

LEMAIRE'S CASE.[22]—A woman aged 25 first suffered from lumbago, backache, pain in the lower extremities and fever. The diagnosis was grip. Five days after the onset, severe headache developed, which resisted medication. Three weeks later diplopia and strabismus were noted, and the eyegrounds showed bilateral congestion of the optic disks. The diplopia was homonymous. The diagnosis was established by lumbar puncture. All serologic tests were tried, but only that for melitococcus gave positive results. (According to Rutherford, this is the first recorded case of papilledema in undulant fever.)

LUNDSGAARD'S CASE.[23]—A farmer aged 64 had a herd of cattle which was attacked with contagious abortion. He stated he had not drunk unboiled milk

19. Cannavò, F.: Complications ocularis de la fièvre ondulante, Rev. prat. d. mal. d. pays chauds **15**:424, 1935.

20. Cohen Boulakia, S.: Neuro-rétino-choroïdite d'origine mélitococcique, Ann. d'ocul. **163**:702, 1926.

21. Godwin, D. E.: Optic Neuritis in Malta Fever, Am. J. Ophth. **12**:747, 1929.

22. Lemaire, G.: Méningite à mélitocoques: Altérations importantes du liquide céphalo-rachidien; hyperglycorachie; guérison, Bull. et mém. Soc. méd. d. hôp. de Paris **48**:1636, 1924.

23. Lundsgaard, K. K. K.: A Case of Iridocyclitis in Association with Febris Undulans Nostra, Acta ophth. **6**:408 1928.

but had taken some unboiled cream. His symptoms were cough and fever and, a fortnight later, a painful left eye. On examination he was found to have marked iridocyclitis with vascularization of the iris. There were interlacing streaks in the lower part of the cornea, reminding Lundsgaard of a type of iridocyclitis with parenchymatous keratitis which he had seen in association with parotitis. The results of all serologic examinations were negative, but the agglutination test for Br. abortus was positive.

Levy and Uzan's Case.[24]—During the second month of undulant fever, meningitis with paralysis of the sixth, seventh and tenth pairs of nerves made their appearance. Ophthalmoscopic examination showed bilateral papilledema. The paralyzed muscle of the eye improved, and eventually there was complete subsidence of the papilledema.

Aubaret and Guillot's Case.[25]—In a case of Mediterranean fever with ocular complications the field of vision was narrowed concentrically. Both optic nerves showed edema. Recovery took place without any special local therapy.

Madievskaya's Case.[26]—A girl aged 13 years had a bilateral ocular condition, characterized by photophobia, lacrimation and pain. She had had a general illness, characterized by fever, malaise, headache and loss of appetite, which began nine months prior to the onset of the ocular symptoms. Malaria was suspected, but no plasmodia were found. In spite of this, she was put on antimalarial treatment, with progressive deterioration in the ocular condition and lowering of vision to 1/10. Ocular examination several months later showed evidence of subacute iritis with synechiae and deposits on the posterior surface of the cornea. The vitreous was clouded. The optic disk was yellowish gray, and the vessels were dilated and tortuous. The patient had a complete medical study, the results of which were wholly negative except for a positive reaction to tests for the presence of Micrococcus melitensis and Br. abortus. The author concluded that this was a true case of undulant fever with complications in the form of iridocyclitis and neuroretinitis of both eyes.

Roger's Cases.[27]—Two cases of paresis of the external ocular motor nerve in the course of melitococcic meningitis are reported.

Villard, Viallefont and Temple's Case.[28]—A man aged 33 complained of rapid and progressive deterioration of vision. He gave a positive reaction to a seroagglutination test for undulant fever. The temperature was high, but the fever yielded to the first injection of melitococcic vaccine. Ocular examination showed a white papilla, small vessels, myopia of 10 diopters and discrete myopic chorioretinitis in the right eye. Vision was reduced to the counting of fingers at 10 cm. The vision rapidly deteriorated to 1/10 in the left eye.

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24. Levy and Uzan, cited by Cohen Boulakia.[20]

25. Aubaret, E., and Guillot: Névrite optique infectieuse bilatérale au cours d'une fièvre exanthématique du littoral méditerranéen, Bull. et mém. Soc. franç. d'opht. **46**:402, 1933; Zentralbl. f. d. ges. Ophth. **31**:492, 1934.

26. Madievskaya, E. Y.: Ocular Complications of Undulant Fever, Sovet. vestnik oftal. **6**:183, 1935.

27. Roger, H.: Les complications méningées de la mélitococcie, Paris méd. **2**:257, 1932.

28. Villard, H.; Viallefont, H., and Temple, J.: Complication rare de la fièvre de Malte: Syndrome tabétique et névrite rétro-bulbaire avec hypertension artérielle rétinienne, Arch. Soc. d. sc. méd. et biol. de Montpellier **14**:224 (Feb.) 1933.

The author's summary follows: "In a subject of 33 years a characteristic undulant fever provoked after a few months a diminution of visual acuity ending in a quasi blindness with contraction of the visual field, with evidences of retinal angiosclerosis. Neurologic examination revealed an abolition of reflexes and disturbances of gait which suggested tabes. The cerebrospinal fluid showed turbidity."

RIZZO'S CASE.[29]—A youth aged 20 had been ill for four months. The symptoms were weakness and numbness; later chilly sensations developed with fever, which assumed the course of a continuous fever with remissions in the morning. Diplopia was noticed one month after the illness began. The serologic tests were negative for typhoid, paratyphoid and syphilis, but were positive for undulant fever. Ocular examination showed the right pupil to be wider than the left, the reaction was sluggish, and there was paresis of the right external rectus muscle. Vision was 1/50, eccentric. There was papillary swelling of the optic disk, and the vessels were normal in caliber and course. The visual field of the left eye was restricted markedly and concentrically for white and colors. There was a central scotoma for red and green. Vision was 1/10. The optic papilla was slightly edematous, with blurred edges and swollen veins. The retina surrounding the disk was edematous.

MOSSA'S CASE.[30]—A woman aged 30 became ill, the symptoms being a sense of debility, lack of appetite and emaciation. When she was seen on March 9, 1934, each eye was completely blind. There was paresis of the abducens nerve on one side. The pupils were slightly dilated and did not react to light. The diagnosis was blindness due to lesion of the primary optic centers. The results of roentgen examination of the head were indeterminate. The patient recovered from the Malta fever, but the optic disks remained completely atrophic. The final diagnosis was blindness due to retrobulbar neuritis caused by Malta fever.

DE JONG'S CASE.[31]—A farmer aged 27 suffered from weakness, fatigue, frontal headache, generalized aches and pains, constipation, nervousness, anorexia, urinary frequency and nocturia. He had a temperature which ranged from 100 to 102 F., and he perspired excessively. He stated that some of the cattle in his neighborhood had been infected with contagious abortion and that his own cow had aborted. Agglutination tests of the blood for the organisms of the Brucella group were as follows: Br. abortus with a dilution of 1:320; Br. suis with a dilution of 1:320, and Br. melitensis with a dilution of 1:120. The phagocytic action of the patient's polymorphonuclear cells on Br. abortus was marked in 94 per cent of the cells and moderate in 6 per cent. He improved without treatment. It was believed that the active stage of the disease was nearly at an end.

The patient was readmitted to the hospital five months later complaining of headache, vertigo, nausea and vomiting. The left pupil was larger than the right, with bilateral weakness of the external rectus muscles. There was neuroretinitis, with blurring of the margins of the disk, slight elevation of the nasal half of the disk and five capillary hemorrhages bilaterally. He was given polyvalent serum

29. Rizzo, A.: Nevrite ottica nel corso di una febbre di Malta, Riv. san. siciliana **24**:1352, 1936.

30. This case was reported at a meeting of the Italian Ophthalmic Society in 1934.

31. De Jong, R. M.: Central Nervous System Involvement in Undulant Fever, with Report of a Case and Survey of the Literature, J. Nerv. & Ment. Dis. **83**: 430, 1936.

made up of the organisms of Br. melitensis and Br. abortus of bovine origin, after which he showed improvement.

Summary: In a 27 year old farmer vertigo, diplopia, tinnitus and vomiting developed eight months after the onset of undulant fever. Clinically he showed neuroretinitis, weakness of ocular motion, nuchal rigidity and tremor of the extremities. The spinal fluid showed a pleocytosis of from 63 to 200, mainly lymphocytes, and an increased protein and a decreased dextrose content. Br. abortus was cultured from the spinal fluid. The patient showed slight improvement after treatment with palliative measures and antiserum.

McCullagh and Clodfelter's Case.[32]—A boy 13 years old had an influenza-like infection. After returning to school a severe headache developed, and he could not see well. Both optic disks were choked; the right eye was blind, and there was little vision in the left eye. A right subtemporal decompression was done. The fever persisted, and the agglutination test for undulant fever was positive. The patient made a slow recovery, with restoration of sight. He lived on a farm and consumed a great quantity of raw milk.

Rutherford's Cases.[33]—Sixty-three patients with undulant fever were examined ophthalmoscopically; 3 showed changes in the disk.

Case 1.—Ophthalmoscopic examination of the patient in this case showed bilateral papilledema, normal visual acuity and contraction of the right field nasally and of the left field concentrically. The clinical diagnosis was encephalitis. Death occurred suddenly fourteen months after the patient's admission to the hospital. The agglutinin test with postmortem blood was positive for undulant fever.

Case 2.—The patient was observed ten months after the beginning of symptoms, which included tingling sensations of the right foot, which spread upward to involving the entire right side and were followed by headache. Subsequent attacks occurred, first on one side and then on the other. There were attacks of unconsciousness. The headaches were accompanied by blurred vision and diplopia. The spinal fluid was under increased pressure, was cloudy and contained a few extracellular gram-negative organisms. Examination at the Johns Hopkins Hospital showed organisms resembling those of undulant fever. These were grown from the spinal fluid. Ophthalmoscopic examination showed the margins of the disks to be blurred, a hemorrhage above the right disk and numerous preretinal hemorrhages. The left fundus showed large hemorrhages and a preretinal hemorrhage. The clinical diagnosis of undulant fever was supported by laboratory tests. The patient died ten months after the beginning of his illness. The microscopic diagnosis was meningoencephalitis due to Br. melitensis variety suis.

Case 3.—Ophthalmoscopic examination showed normal central visual acuity and papilledema of 0.5 diopter in each eye. Later the papilledema increased in each eye. At the Mayo Clinic a parietotemporal exploration was made. No pathologic changes were found. Agglutination tests for undulant fever were positive. A final diagnosis was made of melitensis meningoencephalitis with residual hemiplegia. Rutherford commented: "Melitensis infection of the central nervous system occurs occasionally with or without ocular complications. In

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32. McCullagh, E. P., and Clodfelter, H. M.: Encephalitis Due to Undulant Fever: Report of Four Cases, Ann. Int. Med. **10**:1508, 1937.

33. Rutherford, C. W.: Papilledema in Undulant Fever, J. A. M. A. **104**: 1490 (April 27) 1935.

three of sixty-three cases of undulant fever there were bilateral papilledema, an increase in the spinal fluid pressure, mononuclear pleocytosis, and evidence of infection of the central nervous system by some variety of the melitensis organism. . . . Papilledema is occasionally found in patients in whom the symptomatology is indefinite and leading signs are absent. It is in such cases that undulant fever should be considered in the differential diagnosis."

Carranza's Case.[34]—A boy aged 10 years had an illness of two months' duration. He was admitted to the hospital in a state of coma with spastic contraction of the right leg. The pupils contracted and were reactionless to light. On emergence from the coma the boy complained of seeing green lights. The disks were congested, with blurred margins. The diagnosis was undulant fever. The cutaneous test elicited a positive reaction. Treatment with high voltage roentgen therapy was given. The patient recovered.

Roger's Case.[35]—A woman 28 years of age when seven and a half months pregnant had an intermittent fever. After a premature delivery the fever recurred and lasted four and one half months. She was convalescent for two months. She then had severe headache, nausea and vertigo. There was diplopia due to paralysis of the left external rectus muscle. Eight days later there was paralysis of the right external rectus muscle. The patient recovered. Melitococcic serum was used.

Sella's Case.[36]—A man of 21 had symptoms of meningitis. On admission to the hospital ten days after his illness began he had dilated pupils. Bilateral optic neuritis was found. Complete recovery followed the intravenous administration of specific vaccines.

COMMENT

It will be noted that most ocular lesions occurring in the course of brucellosis do not destroy the integrity of the globe. Hence enucleation is not demanded. This fact explains the lack of any pathologic examination of a human eye that has been affected with brucellosis. Some interest therefore attaches to Orloff's investigation of the globes of guinea pigs which died of melitensis.

Orloff[37] examined the eyes of 19 guinea pigs ill with melitensis. Some presented ocular lesions in life. At the first sign of ocular participation circumcorneal injection was noted. There was slight central clouding of the cornea. The iris was more or less hyperemic; the pupil was occupied by a grayish exudate. Microscopic examination of the cornea showed in cases of slight involvement the following alterations: extensive desquamation of the epithelium in the central part, epithelial degeneration and formations of vacuoles in the nuclei at the periphery, formation of vacuoles in the peripheral endothelium and

34. Carranza, J. P.: Meningoencefalitis aguda, difusa melitocócica, Crón. méd., Lima **53**:281, 1936.

35. Roger, H.: Les complications cérébrales de la mélitococcie, Marseille-méd. **2**:591, 1929.

36. Sella, M.: Meningite acuta da infezione melitense: Efficacia del trattamento vaccinico per via endovenosa, Gior. di clin. med. **16**:466, 1935.

37. Orloff, K. C.: Melitokokkia (Malta Fieber) und Auge, Klin. Monatsbl. f. Augenh. **81**:582, 1928.

grave lesions of the endothelial cells, corresponding to the central clouding. In several cases the corneal lesions were much more pronounced, and the microscopic picture acquired a great similarity to that of syphilitic parenchymatous keratitis. The iris in the majority of cases was diffusely infiltrated, and there were numerous posterior synechiae.

The ciliary body appeared to be most affected; in all cases there was an infiltration of lymphoid and epithelioid cells. The vitreous was often fluid. In about half the cases the lens showed definite signs of inflammatory cataract. In the anterior portion of the choroid there were found hyperemia and here and there foci of infiltration. In the posterior portion of the uvea there was migration of the subretinal pigment with round cells and fibrin.

On the basis of these findings, Orloff stressed the practical importance of infection of the eye with melitensis, which clinically as well as pathologically seems to have a great similarity to ocular tuberculosis.

An immense amount of work has been done on brucellosis in the past twenty years. Those who have undertaken the study of the disease either in the laboratory or clinically have become enthusiastic about the possibilities and implications of this new malady; in fact, one is inclined to feel that some authors have allowed their enthusiasm to outrun their judgment. Evidence is accumulating, however, that some ocular maladies hitherto ascribed to other origins may be caused by brucellosis. The external ocular muscles, the cornea, the uveal tract, the retina and the optic nerve have all proved vulnerable. Who has not been thwarted in his most painstaking efforts to establish the cause of a chronic uveitis? Should not the ophthalmologist include in his list of possible etiologic factors a disease that is widespread and one which has been proved to be capable of affecting almost every tissue of the body?

Charles Nicolle, one of the most distinguished students of undulant fever, has made this prediction concerning the disease:

> Mediterranean fever is in the course of evolution, and is tending to become chronic. It is a malady which because of its manifestations and chronicity will become one of the commonest and most stubborn diseases. . . . Mediterranean fever is a disease of the future.

3720 Washington Avenue.

DISCUSSION

Dr. Arthur J. Bedell, Albany, N. Y.: Dr. Green has thoroughly covered the subject of the ocular evidence of undulant fever. I would, however, sound a note of warning, and that is not to ascribe all the ocular signs present in a patient who has undulant fever to that condition, until all others have been excluded.

To illustrate this, a man aged 39 had a trivial injury to his right eye. This was so slight that he paid no attention to it and did not see a physician. Four days later he covered his uninjured eye and found that he could not see from the injured one.

A photograph taken within a few days showed a white optic nerve. The macular region was dark, and surrounding the pale disk were concentric white rings, ruptures in the choroid. The vision was 10/200.

The patient's attorney introduced questionable evidence to the effect that the slight conjunctival injury caused a localization of the organisms of undulant fever in the back part of the eye. The man was awarded compensation for the loss of the vision of his right eye.

The point is that a patient may carry organisms of many kinds. I do not believe that there is justification for considering choroiditis the result of undulant fever simply because the patient has that infection. Undulant fever and its effect on the eyes are open to further investigation.

Dr. Albert C. Snell, Rochester N. Y.: I wish to report the case of an 8 year old child in whom central retinitis developed. Roentgenograms of the chest were normal, and cutaneous tests for tuberculosis gave negative results. In trying to find some cause for the condition, I found that undulant fever was present in a small, private dairy. The owners of the dairy maintained their own herd of five or six Jersey cattle, and the veterinarian proved that the herd was infected. The patient had some recurrent fever which resembled malaria, but there was no malaria in that particular part of the country, so that the diagnosis was made of brucellosis in the cattle and of undulant fever in the child. Typical retinitis was present for some time, with considerable elevation of the fundus around the macular region. The central vision was lost.

Dr. Eugene M. Blake, New Haven, Conn.: I wish to add 1 case to Dr. Green's series. The patient, a woman of 55 years, was seen in June 1936 at the New Haven Hospital. The onset of her illness occurred in February of the same year. Following the extraction of a tooth she had had frequent sore throats, and the cervical glands were tender and swollen. On April 24 she had a fairly severe chill. At the time of her entrance to the hospital the temperature was 103 F., the entire pharyngeal wall was red and the right wall was bulging.

Laboratory examination revealed 5,400,000 red blood cells and 9,300 white blood cells, with 60 per cent polymorphonuclear leukocytes, 27 per cent lymphocytes, 11 per cent large mononuclears and 1 per cent eosinophils. The culture from the throat yielded Streptococcus viridans (4 +), Pneumococcus (1 +), Haemophilus influenza (1 +) and Micrococcus catarrhalis (1 +). The cutaneous reaction for undulant fever was 4 +.

One transfusion of 500 cc. of citrated blood was given.

The patient remained in the hospital for seven weeks, and during the entire time there was a daily rise of temperature to over 100 F. She had been mildly diabetic for twenty years, and this condition was readily controlled by small doses of insulin and diet.

In 1934, while sojourning in Florida, the patient had what was diagnosed as a dendritic ulcer, which healed, leaving a small scar on the

right cornea. Soon after admission to the hospital, in 1936, the right eye became inflamed, and a small epithelial defect appeared at the upper margin. In spite of treatment, this increased until two thirds of the cornea stained with fluorescein. This gradually healed, but the cornea became deeply infiltrated and has remained so to date. There are anesthesia of the cornea and a yellowish white infiltration, which barely allows the pupil to be seen through it. There are deep and superficial vessels, and the condition in the cornea resembles severe interstitial keratitis more than anything else. In spite of the use of medicaments, such as ethylmorphine hydrochloride, yellow mercuric oxide ointment and heat, the condition remains unchanged. There is a secondary glaucoma, with tension of 36 mm. (Schiötz), which is fairly well controlled with a 1 per cent solution of pilocarpine hydrochloride.

It is, of course, entirely questionable whether the corneal disease is definitely related to the undulant fever or was simply a result of the streptococcic infection. However, the corneal picture is unusual and should be added to the conditions complicating undulant fever, along with those reported by Dr. Green, until a clearer understanding of the disease is obtained.

Dr. John Green, St. Louis: My primary purpose in writing this paper was to make ophthalmologists "brucellosis conscious." In St. Louis one of the large laboratories is making it a practice to carry out cutaneous tests on practically all patients who are sent in for Wassermann or tuberculin tests, and in that way this one laboratory has found 500 cases of infection with Brucella abortus within two years. It does seem as though there is a great difference in the prevalence of the disease in different parts of the country. In some of the great dairy states, such as Iowa and Michigan, more studies on undulant fever have been made than in other parts of the country, and in New York also there have been a number of excellent studies. On the basis of present knowledge it is impossible to be certain in many cases of undulant fever in which there are ocular lesions as to the causative relation to the organism, but with all other tests negative and the reaction of the test for brucellosis strongly positive, the relationship is suggestive.

The complication in the case reported by Dr. Blake is very rare. Judging from the literature, the cornea is about the least involved of all the ocular tissues.

MALIGNANT MELANOMA OF THE CHOROID WITH METASTASES

REPORT OF A CASE

FORREST J. PINKERTON, M.D.
HONOLULU, HAWAII

The comparatively rare condition reported here has been called melanosarcoma and is generally referred to in this manner in textbooks, but recently many authors have preferred to call it malignant melanoma, as there is some doubt as to its exact origin.

REPORT OF CASE

A stalwart man 26 years of age, of Portuguese parentage, who first consulted me on Aug. 16, 1935, gave a history of severe pain in the left eye of two weeks' duration. He had noted a gradual reduction of vision in the left eye several weeks prior to the onset of acute pain, and for several weeks before consulting me he had noticed from time to time a dull ache in the left eye. The patient said that he had not received any injury and could not connect the symptoms of the left eye with any accident or recent acute inflammation.

Examination revealed the vision of the right eye to be 20/20, while that of the left was reduced to the doubtful counting of fingers in the temporal field. The right eye was essentially normal in all particulars. The fundus was clear, and no evidence of a pathologic process was noted. The tension of the right eye was 20 mm. of mercury (McLean). The left eye, however, showed marked, deep, pericorneal injection; the cornea was quite steamy, and the anterior chamber was extremely cloudy and shallow. The tension of the left eye was 100 mm. of mercury (McLean). The iris could be seen with difficulty. The pupil was slightly dilated; and the picture in general resembled that of an acute inflammatory glaucoma. Details of the fundus could not be made out.

The patient was hospitalized, and conservative treatment was tried for three days without success.

On August 29 posterior sclerotomy of the left eyeball was performed between the superior rectus and the external rectus muscle and posterior to the ciliary body. There immediately presented in the wound a large herniating mass of dark red or black material which resembled clotted blood. The excess amount of this material was expressed and sent to the laboratory for microscopic examination. The laboratory report stated that the condition was a melanoma, and on August 31 the left eyeball was enucleated and sent to the laboratory. The pathologic examination showed a mass of friable, dark colored tissue nearly 1 cm. in thickness which appeared to have its origin in the choroid in the temporal portion of the eye. The pathologic diagnosis was melanosarcoma or malignant melanoma.

A thorough general physical examination, including urinalysis and a Wassermann test of the blood, revealed no evidence of any abnormality. The patient was an unusually well developed physical specimen. The socket appeared clear, and his recovery was uneventful so far as the enucleation was concerned. The

right eye was carefully studied with a slit lamp and the Gullstrand ophthalmoscope, and no evidence of a pathologic process could be found. The patient was kept under observation until December 1935, having received several doses of high voltage roentgen therapy to the left orbital region in the interim.

He was allowed to return to his home on one of the other islands with instructions to report at regular monthly intervals for further observation. This he failed to do, and he was not seen again until July 1936, when he returned complaining of being unable to keep the artificial eye in place owing to a swelling of the left orbital tissues. Examination revealed a large mass of tissue which could be seen through the transparent conjunctiva, herniating forward so that an artificial eye could not be kept between the lids. A small incision was made in the mass, and some of the tissue was sent to the laboratory, where a diagnosis of melanosarcoma was made.

After consultation, the patient permitted complete exenteration of the left orbit. This was done radically, electrocoagulation being used to control the bleeding points. Large doses of high voltage roentgen irradiation and radium were given, and recovery was uneventful. The orbit remained free from any recurrence of the growth. The patient was observed daily for dressings, and with the exception of some yellow serosanguineous discharge, the left orbit remained free from any recurrence of the disease until his death.

In March 1937 he was again admitted to the hospital for the purpose of repairing the left orbit by application of skin grafts, several pieces of skin being removed from the inner part of the arm and transplanted into the orbit, with successful results. With the exception of a small fistulous process in the nasal side over the lamina papyracea, the left orbit was satisfactorily epithelized. Ultimately, the orbit became clean and required nothing more than a light pad of gauze from day to day. The patient, however, began to complain of loss of weight and some vague abdominal pain, which he thought was due to dietary indiscretion. Metastasis was suspected, although up to this time no evidence of metastatic involvement had been found, in spite of careful observation. The patient was again referred to an internist, who thoroughly examined him and could find no evidence of a metastatic process. He continued to lose weight, and in June 1937 had lost a total of approximately 40 pounds (18 Kg.) during the preceding six months. Physical examination now showed some tenderness over the liver and slight enlargement below the costal margin. The hemoglobin content was 72 per cent at this time. The patient was given large doses of roentgen irradiation to the area of the liver but continued to complain more and more severely of abdominal pain. The liver continued to enlarge, and by August 8 it was enlarged 4 fingerbreadths below the costal margin. His condition seemed so hopeless that his family felt they would like to have him return to his home, which he was permitted to do. He died on Oct. 2, 1937.

HIGHER VISIBILITY IN A ROENTGENOGRAM ILLUMINATOR

C. E. FERREE, Ph.D.

AND

G. RAND, Ph.D.

BALTIMORE

The usefulness of roentgen plates and films to the surgeon sustains a direct, and to the diagnostician in many cases perhaps a proportionate, relation to their visibility. Important factors in attaining a high degree of visibility are the brightness and color relationship of the background to the details to be discriminated, and the intensity of the illumination. With respect to the first of these factors, it may be noted that details neutral as to color are best seen on a clear white background. With respect to the second factor, it may be said that the amount of light needed to give the highest power to discriminate detail varies with the density of the film and with the person making the examination. Not all films or all parts of the same film are of the same density; and the intensity of light giving the highest visibility varies widely from person to person, depending on such factors as age, size of pupil, refractive condition of the eyes, susceptibility to glare, clearness of the media, the condition of the sensorium, etc. If too little light is used, low visibility occurs from insufficient stimulation of the retina; if too much is used, glare and harmful scatter of light are produced. From film to film there is variability in both absorption and scattering action. All these factors lower visibility and confuse the discrimination of detail.

In figures 1 and 2 is shown a roentgenogram illuminator which was devised to satisfy the foregoing requirements for high visibility. With it, the intensity of light may be varied in continuous series from zero to full without change in the color or composition of the light or in the size, shape or location of the illuminated area. This variation is accomplished also without any change in the position of the lamp.

The device for producing the variable illumination consists essentially of a reflecting housing, which may be of any size or shape desired, a rotatable shutter and a diffusing element to break up all shadows and give an evenly distributed and well diffused illumination. From this it can be seen that the construction can readily be adapted to any size or type of lamp and to cover any range of intensity that might be desired.

From the Research Laboratory of Physiological Optics.

The unit further consists of a flaring bonnet-like construction on the front of the housing. Across the opening of the housing just in front of the shutter is a plate of etched Whiterlite glass, which serves to give both color correction and in part the diffusion mentioned previously. Approximately 5 inches (13 cm.) in front of this glass and across the opening of the bonnet-like extension is a sheet of Celestialite glass, which serves to complete the diffusion and to give a transilluminated background against which the plate or film is viewed. So good is the diffusion thus afforded that no trace of shadow from the vanes

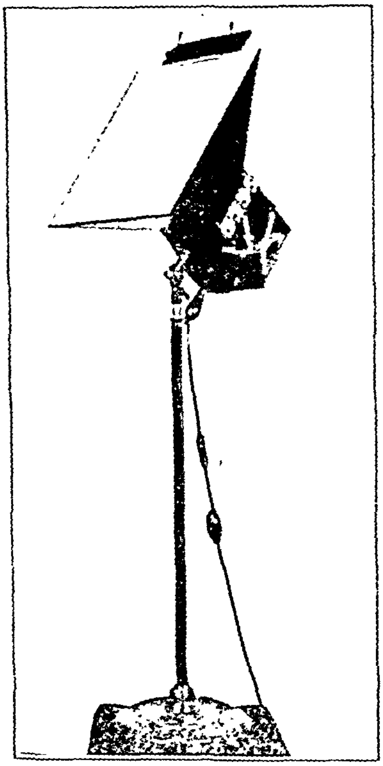

Fig. 1.—A roentgenogram illuminator with variable intensity of light, corrected for color.

can be seen on this glass. At the top of the extension a clip is added to hold the plate or film in position. For viewing plates or films of different sizes, masks are provided having differently sized apertures. Depending on the amount of color correction that is wanted, Whiterlite or Daylight glass may be used for the plate directly in front of the shutter. Our experience is that the better the color correction, the better the results. With the diffusion given with this setup and with sufficient color correction and the proper adjustment of intensity, a greatly improved visibility is obtained. Quite good results can be obtained, however, by making both plates of Celestialite glass. This

glass, it may be noted, affords both an excellent degree of diffusion and some color correction.[1]

The means for varying intensity consists of four vanes, which extend across the opening of the housing in such relation to each other that when their flat surfaces are parallel to the beam of light the maximum amount of light passes through the opening, and when they are rotated to a position at right angles to the beam the light changes in continuous series from full intensity to zero. In changing the intensity, the vanes are so actuated as to cause the contiguous vanes to turn in opposite directions. This insures an absence of shift in the position of the illuminated area and of change in its size and shape. When all the vanes move in the same direction, as is the case with Venetian blinds,

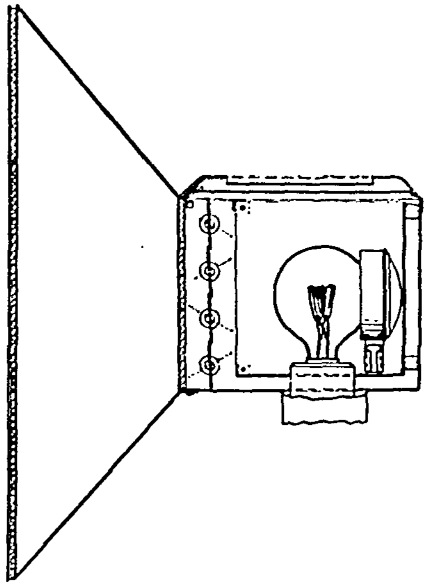

Fig. 2.—A vertical section of the roentgenogram illuminator, showing the shutter for varying the intensity of light.

all these changes take place. In the various models of the instrument different means of actuating the vanes have been employed. In the unit shown in figure 1, a pin or shank which passes through an opening to the outside of the housing is attached at the central point of each end of the vane. Mounted at the outer end of these pins on one side of the unit are cogwheels or gearings, 1½ inches (3.8 cm.) in diameter, which mesh with each other. On one of these cogwheels is a small knob for convenience in turning the vanes. In one of the latest models a simpler means has been devised which lies within the housing and is

1. Whiterlite glass and Daylight glass may be obtained from the Macbeth Daylighting Co., New York, and Celestialite glass from the Gleason-Tiebout Co., New York.

entirely free from slack or backlash. This is in the form of a thin plate with a central longitudinal slot, through which pass the axle pins at the ends of the vanes. This slot permits the plate to travel back and forth as the vanes turn. Above and below it in alternate sequence are short vertical slots which engage pins suitably positioned at the ends of the vanes. To the end of the axle of one of the vanes is attached a knob. When this knob is turned, the contiguous vanes rotate in opposite directions as is desired.

When there is need of varying the intensity of light, it is the tendency among manufacturers, for purely commercial reasons, to use a rheostat. There are many objections, however, to this means of varying the intensity of light, particularly in the present case. In fact, the mechanical means described here was devised to get away from these objectionable features. The correct use of a rheostat in relation to a source of light is, in conjunction with an ammeter or a voltmeter, to guard against variations in the intensity of light, not to produce them.

Several years of hospital experience has led us to believe that high visibility in roentgenograms is greatly prized by surgeons and diagnosticians and that even small improvements in this direction merit serious consideration. We would especially emphasize here the need of having just the right amount of light on the roentgenogram and the impossibility of getting this amount for different roentgenograms and different parts of the same roentgenogram unless the change of intensity can be accomplished in continuous series. This could not very well be done, for example, with a rheostat. The difference between the most favorable intensity and the intensity which gives either simple or veiling glare or both is in some cases and for some persons quite small. In this connection we would call to mind the wide range of variation there is for different persons in the amount of light that is most favorable for the discrimination of details and in the difference between this amount and the threshold of glare. In both of these respects age is a most important factor. In general, in the adaptation of intensity to the needs of the individual the difference in the requirements of those under and those above 40 years of age is in many cases astonishingly great. Differences in the scattering action of the film also create a need for a means of varying intensity of light in continuous series or by small steps.

We would further strongly emphasize that the change of intensity should not add color to the background against which the details of the roentgenogram must be viewed. Both the film and the light are already too colored. Details neutral as to color cannot, all must agree, be seen clearly against a colored background, particularly when that color is produced by transillumination. The use of a rheostat, for example,

would normally mean that the lamp would be run at a reduced voltage, which would cause a change in color toward the long wavelengths. This change of color might in some cases offset almost if not entirely the benefit obtained from change of intensity. The transmitted light is already too yellow. Its further change toward orange by the use of a rheostat is bad indeed. In addition, change in the composition of light changes the amount of scatter given to the light in passing through the film. The long wavelengths are scattered more than the short.

In any attempt to improve the visibility of details in the roentgenogram, color correction of the light is vastly important as an initial step. The ideal condition is to start with light of daylight quality and to vary the intensity by just the right amount to give highest possible visibility without changing the color and composition of the light. The full effect of the benefit so obtained must be experienced to be believed. In our opinion no surgeon or diagnostician who has had the opportunity of experiencing these benefits would fail to commend them or hesitate to incur the additional expense of obtaining them, if indeed any considerable additional expense is required. The expense of the device for varying intensity is, we may say, slight in the simplified form. In any event, where human welfare, even human life, are at stake, small differences in cost are in comparison of little consequence. Our motive for devising this improvement has been the experience of an important need.

The model shown in figure 1 was not made to suit any particular situation. It was constructed solely for the purpose of trying out the improvement in visibility. But even in the form shown, it might perhaps be convenient for use in an operating room where the surgeon needs to look at the film while the operation is in progress. The device as described can be readily adapted to any type of stand or cabinet for the examination and study of roentgenograms.

Made after a suitable design, the equipment serves an excellent purpose also for viewing dental films and lantern slides. For the equipment in small sizes, a single rotatable vane would suffice for the control of intensity. Properly designed, we believe that this equipment in its various forms would render a great and much needed service to many persons. In suitable designs and sizes, a magnifying element can also be used, either mounted on the instrument or held in the hand. In addition to its service for viewing roentgenograms, we have personally derived great satisfaction from using the unit in conjunction with a magnifier for studying lantern slides in preparation for lectures and other purposes. Because of the grain of the film, there is, of course, a limitation to the magnification that can be used to advantage. In a magnifying system it may also be noted that a means of varying intensity to suit the degree of magnification is an advantage.

SUMMARY

Important factors in attaining high visibility in roentgen plates and films are the brightness and color relationship of the background to the details to be discriminated and the intensity of illumination. Details neutral as to color are best seen on a clear white background. The amount of light needed to give highest power to discriminate detail varies with the density of the film and the person making the examination. Not all films or all parts of the same film are of the same density; and the intensity of light giving the highest visibility varies widely from person to person, depending on age, size of pupil, refractive condition of the eyes, susceptibility to glare, clearness of the media of the eyes, condition of the sensorium, etc. If too little light is used, low visibility occurs from insufficient stimulation of the retina; if too much is used, glare and harmful scatter of light are produced. A roentgenogram illuminator is described which was devised to satisfy the foregoing requirements for high visibility. By means of a rotatable shutter, the contiguous vanes of which turn in opposite directions, the intensity of light may be varied in continuous series from zero to full and without change in the color or composition of the light. Neither of these important requirements can be satisfied when, e. g., a rheostat is used to vary the intensity. Color correction and high degree of diffusion are secured by filtering the light first through etched Macbeth daylight or Whiterlite glass and second through Celestialite glass. The construction can readily be adapted to any type of stand or cabinet.

VASCULAR OBLITERATION FOR VARIOUS TYPES OF KERATITIS

ITS SIGNIFICANCE REGARDING NUTRITION OF CORNEAL EPITHELIUM

TRYGVE GUNDERSEN, M.D.
BOSTON

Two of the existing concepts of the function of blood vessels in corneal tissues are diametrically opposed. According to the first, the one generally held, the function of the vessels is reparative; according to the second, it is destructive. Growth of blood vessels regularly accompanies healing throughout the body; repair and vascularization go hand in hand in practically all inflammatory processes. A well marked interstitial keratitis rarely shows any regression until from four to eight weeks after the onset, at the time when stromal vessels become visible. The same is true of many other corneal inflammations. For instance, some deep herpetic infections fail to subside for many weeks or even months. During this period of time blood vessels have made their appearance in the corneal stroma, and when healing does occur it is likely to be attributed to the increased vascularity. On the other hand, the clinical course of a fascicular ulcer, "wandering phlyctenule," indicates that the blood vessels actually cause the tissue necrosis and thus activate the ulceration. In this disease one finds slowly creeping across a cornea an ulcer apparently driven by a leash of blood vessels close at its heel. Hence it must be assumed either that corneal vessels have diametrically opposite functions under different conditions or that the interpretation of the function of the blood vessels is at fault in one or the other concept.

Wounds may heal by primary intention with little assistance by blood vessels, as is illustrated in corneal grafting. A full thickness graft usually adheres perfectly and grows solidly into position before any vessels make their appearance. It is clear that vessels are of no assistance in this type of healing; in fact, the postoperative ingrowth of blood vessels is the greatest hazard in keratoplastic operations. The concept that well vascularized organs are less subject to infections than those with a poor blood supply is not borne out in the eye. The

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From the Howe Laboratory of Ophthalmology, Harvard University, and the Massachusetts Eye and Ear Infirmary.

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avascular cornea rarely becomes infected after trauma or during the course of an acute conjunctivitis which represents a severe infection of an adjacent well vascularized tissue. The lens, which is remotely dependent on blood supply for nutrition, if uninjured practically never becomes infected, and similarly the vitreous is very resistant, since prolapses of this tissue into operative wounds remain free from infection even when persisting for many weeks.

The observation that reducing the supply of blood has a healing effect on corneal lesions was first made almost two centuries ago. The use of astringents or of surgical treatment to effect this result was advocated then more universally and with greater clarity than it is today.

Boury,[1] as early as 1743, advised cutting the vessels all around the cornea. He called his operation ophthalmophlebotomy. De Saint Yves [2] (1767) used a similar procedure.

Antonio Scarpa [3] (1801), one of the most astute observers of his day, wrote as follows:

> The treatment that is indicated for a nubecula of the cornea consists in shrinking the varicose vessels of the conjunctiva down to their natural caliber or, if this cannot be accomplished, in severing the trunks of the more prominent varicose vessels of the conjunctiva from their slender roots at the point where the latter leave the external surface of the cornea in the region of the nubecula. The former method of treatment is carried out by means of local astringents and corroborants mentioned in the preceding chapter, first place being given to the ophthalmic ointment of Janin [4]; these remedies suffice provided the nubecula is in its early stage of limited extent. But when it has advanced near the center of the cornea and the relaxation of the conjunctiva and its vessels is considerable, the most effective and quick-acting expedient of all those hitherto proposed is recision of the bundle of varicose veins near its roots, that is, in proximity to the nubecula of the cornea. By means of such recision the blood that has slowed

1. Boury, J. W.: Dissertatio de maculis corneae earumque operatione chirurgica, apotripsi, Lausanne, 1743.

2. de Saint Yves, C.: Nouveau traité des maladies des yeux, Amsterdam, Arkstée & Merkus, 1767, pp. 171 and 178.

3. Scarpa, A.: Saggio di osservazioni e d'esperienze sulle principali malattie degli occhi, ed. 1, Pavia, Baldassare Comino, 1801, p. 106.

4. The ophthalmic ointment of Janin consists of melted hog fat, 1 ounce (31 Gm.); prepared zinc oxide, 2 drachms (7.7 Gm.); Armenian bole, 2 drachms (7.7 Gm.), and ammoniated mercury, 1 drachm (3.8 Gm.). The hog fat must first be washed three times in rose water. The drugs are to be mixed in a glass mortar. The preparation is imperfect and of little value unless the ingredients have been reduced to an impalpable powder. Janin wrote (1772) that this is an excellent ophthalmic remedy, not only for inflammation but for emptying varicose veins and restoring them to their natural state. It dissipates (corneal) maculas, reduces engorgement of the lids, cleanses and cicatrizes ulceration of the margins of the lids and of the cornea. Armenian bole is a compact, soft, unctuous clay from Armenia. It contains ferric oxide and has astringent properties.

up in the roots of the dilated veins on the surface of the cornea is at once drawn off, and the varicose vessels of the conjunctiva more readily regain their normal tone and caliber; at the limbus is formed a kind of filter for the egress of whatever serum or other albuminous material has spread in the tissue of the thin lamina of conjunctiva overlying the cornea or in the cellular tissue that binds together these two membranes. It is really surprising how quickly a nubecula of the cornea is dissipated by means of this operation, for in the majority of cases the lapse of twenty-four hours after the recision of the bundle of varicose vessels of the conjunctiva is sufficient to cause the disappearance of the opacity at the site of the nubecula.

Charles Bell [5] in 1816 stated:

The practice is to extirpate the tortuous fasciculus of vessels, whose elongation over the cornea *caused*, or necessarily accompanied the formation of the opacity, and which we may now suppose feeds and supports it.

Toward the middle of the nineteenth century the *Ophthalmic Hospital Reports* published articles by Bader [6] on syndectomy and by Lawson [7] on peritomy. Bader reported the results of operation in 24 cases of vascularized cornea with or without granular lids. Some of his results were truly remarkable. He concluded: "Syndectomy is a less dangerous and troublesome mode of treatment than that by inoculation of pus." Lawson was not greatly impressed by his results but concluded that in milder forms of vascularized cornea, when inoculation with pus could not be judiciously advised, peritomy might be of benefit.

Furnari,[8] of Paris, while working in Algiers, first performed the operation of peridectomy for trachoma as early as 1842 but did not publish his results until 1862. Although his own results were excellent, the operation was found too radical by other surgeons, and their bad results served to relegate the procedure to oblivion for the rest of that century.

In the present century the operation of syndectomy was revived and modified by Denig.[9] Rather than allow the bulbar conjunctiva to replace itself over the excised area, buccal mucous membrane was grafted on the exposed sclera. This foreign mucosa was said to retard the downgrowth of vessels onto the cornea and to be resistant to trachomatous infection. Seefelder,[10] Friedman [11] and Kaminski [12] have since demonstrated that the graft does become infected if put on during the acute

5. Bell, C.: Operative Surgery, ed. 2, Hartford, Hale & Hosmer, 1816, vol. 2, p. 90.
6. Bader, C.: Ophth. Hosp. Rep. **4**:19, 1863-1865.
7. Lawson, G.: Ophth. Hosp. Rep. **4**:64, 1863-1865.
8. Furnari, S.: Gaz. méd. de Paris **17**:54, 83, 114, 147, 176 and 210, 1862.
9. Denig, R.: Ztschr. f. Augenh. **25**:278, 1911.
10. Seefelder, R.: Klin. Monatsbl. f. Augenh. **81**:68, 1928.
11. Friedman, B.: Rationale of Denig Transplant in Trachoma, with Microscopy of Graft in Two Cases, Arch. Ophth. **4**:868 (Dec.) 1930.
12. Kaminski, D. S.: Klin. Monatsbl. f. Augenh. **87**:60, 1931.

stage. Friedman furthermore stated that the vascularity of pannus is apparently not altered by the removal of the limbal conjunctiva or its replacement by a graft.

Weller,[13] Teale,[14] Ziegler,[15] Fox,[16] Whitehead,[17] Gallewaertz,[18] D'Amico,[19] Alonso [20] and others have written enthusiastic reports about peritomy.

Szokolik [21] reported good results after peridectomy in 12 cases of pannus. He found no need for the transplantation of buccal mucosa and suggested that the deep blood supply was all that was necessary for corneal nutrition. Whitehead advocated peritomy especially for some forms of corneal tuberculosis.

Most textbooks fail to discuss the pathogenesis of wandering corneal ulcers. Their writers are aware that fascicular ulcers occur but rarely attempt an explanation of this curious disease.

D'Amico suggested an explanation for the beneficial effect of partial peritomy in cases of trachomatous pannus by assuming that the uncut blood vessels underwent compensating dilatation, thereby enhancing nutrition and carrying away toxins. To me, this explanation seems inconsistent.

In "Das Kurzes Handbuch der Ophthalmologie" (Schieck and Brückner) [22] the most modern concept of the etiology of "wandering phlyctenules" is explained as follows:

> An explanation for this peculiar condition is possible if we assume that the blood vessels in these cases carry tuberculous antigens. Then the newly formed vessels do not bring about the desired healing but an anaphylactic inflammation in the sensitized cornea, which causes the ulcer to continue unabated. This argument is strengthened by the observation that in certain cases when the vessels are divided at the limbus the inflammation subsides. Just as welcome as a pannus is in scrofulus keratitis, just so destructive are the vessels in "Gefassbandchen."

This allergic reaction may occur in patients with phlyctenular keratitis who are very sensitive to tuberculin, but it seems unreasonable to assume

13. Weller, C.: Die Krankheiten des menschlichen Auges, Berlin, Schüppel, 1826, p. 208.

14. Teale, T. P.: Tr. Ophth. Soc. U. Kingdom **21**:146, 1901.

15. Ziegler, S. L.: The Surgery of Trachoma, J. A. M. A. **30**:131 (Jan. 15) 1898.

16. Fox, L. W.: Ann. Ophth. **12**:614, 1903.

17. Whitehead, A. L.: Brit. J. Ophth. **6**:529, 1922.

18. Gallewaertz, M.: Ann. d'ocul. **160**:342, 1923.

19. D'Amico, D.: Cong. Soc. ital. d'opht. **10**:22, 1930.

20. Alonso, A. F.: La peritomia en las ulceras cronicas vasculares de la cornea, Internat. Cong. Ophth. **1**:371, 1922.

21. Szokolik, E.: Klin. Monatsbl. f. Augenh. **78**:693, 1927.

22. Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1931, vol. 4.

that it is always tuberculous antigen that causes a "wandering ulcer" at the site of dendritic keratitis (cases 1 and 2) or an ulcer in the vascularized scar of an old corneal laceration (case 24). In the majority of cases it would seem that it is some noxious substance other than tuberculous antigen that is brought in by the blood vessels.

Where does the cornea get its nutrition? The generally accepted notion given in textbooks is that nutriments enter by a process of diffusion from the pericorneal blood vessels, but that they may also enter from the aqueous. Apparently this view is based on the experiments of La Queur [23] (1872) and Gruber [24] (1894), who after producing rust spots in the corneas of cats and injecting potassium ferrocyanide into the circulation noted that the spots became blue from the peripheries toward the centers of the corneas. The same effects were noted after injecting ferrocyanide into the anterior chambers. The cornea seems to be able to live and remain transparent when either source of nourishment alone is cut off, as illustrated by Gruber in doing complete peritomy and by Coccius [25] (1852) in keeping the anterior chamber filled with air. The cornea is said to have an exceedingly "slow metabolism" (Duke-Elder [26]) which enables it to be satisfied with nourishment from either the aqueous or the pericorneal vessels. There are no direct measurements to support the contention that its metabolism is slow. On the contrary, Bessey [27] is finding that excised corneas of rats show an astonishingly fast metabolic rate. Working with a Warburg respirator, which measures accurately the oxygen consumption of tissues, he found that the entire cornea per milligram of dry weight utilizes about 4 cu. mm. of oxygen per hour, i. e., four-sevenths as much as liver tissue and four-sixths as much as muscle tissue, which have rapid respiratory rates, and four times as much as cartilage. In view of the fact that the bulk of the entire cornea consists of its stroma, a tissue somewhat like cartilage, the high respiratory rate of cornea found by Bessey points toward a rapid respiration of the epithelium itself. The most active portion of the cornea certainly is the epithelium. Its tremendous regenerative power is known from clinical experience. No wounds heal as rapidly as do those of the corneal epithelium. The epithelium may regenerate over the entire surface of the cornea in three or four days.[28]

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23. Laqueur, L.: Centralbl. f. d. med. Wissensch. **10**:577, 1872.

24. Gruber, R.: Arch. f. Ophth. **40**:25, 1894.

25. Coccius, E. A.: Ueber die Ernährungsweise der Hornhaut und die Serumführnden Gefässe im menschlichen Körper, Leipzig, I. Müller, 1852.

26. Duke-Elder, W. S.: Textbook of Ophthalmology, St. Louis, C. V. Mosby Company, 1933, vol. 1.

27. Bessey, O. A.: Personal communication to the author.

28. Gundersen, T.: Herpes Corneae with Special Reference to Its Treatment with Strong Solution of Iodine, Arch. Ophth. **15**:225 (Feb.) 1936.

Surely an active tissue like corneal epithelium must require a ready source of nourishment as well as oxygen. It is not yet possible to say with certainty whence they come. The oxygen necessary to support corneal respiration is readily available if the cornea can utilize the surrounding atmosphere. The cornea of an excised eye placed in an atmosphere saturated with moisture will remain transparent for twenty-four hours, but if the air is replaced by hydrogen, the cornea rapidly becomes opaque (Bullot,[29] 1904). Fisher [30] (1930) has shown that oxygen can pass only from the epithelium toward the anterior chamber and carbon dioxide only in the reverse way.

To determine if corneal tissue could live in aqueous alone, I devised the following experiment: A full thickness piece of cornea was removed from the eye of an animal and transferred to the anterior chamber of the opposite eye. This experiment was carried out on 17 cats and 10 rabbits. Trephine disks 3.5 mm. in diameter were implanted in the eyes of 24 animals, and strips of cornea 3 by 10 mm. were implanted in the eyes of 3 animals. In all except 1 instance the implants adhered to the iris and became vascularized. In the 1 instance in which this complication did not occur, the implant was free in the aqueous at all times, and it remained so for twenty months without ever becoming adherent to the iris. Thus it was possible to observe the changes which occurred in corneal tissue surrounded by normal aqueous alone. The corneal disk remained relatively transparent for about six months and diminished little if any in size. It then became semi-opaque and globular and remained so without any visible change for the remaining fourteen months. The eye was then removed and the disk studied histologically in serial sections. The epithelium had entirely disappeared. The stroma showed no necrosis, hyalinization or fibrosis, while the endothelium had proliferated and surrounded the entire piece. It is noteworthy that even in the other experiments, though the implants became vascularized through their adhesions to the iris or cornea, the epithelium was found absent on microscopic examination. The details of this experiment have been reported elsewhere.[31]

This direct experiment shows that the stroma and "endothelium" can live for a prolonged period in aqueous alone but that the epithelium cannot.

On first thought, this statement may seem to conflict with the fact that epithelium can grow in the anterior chamber, as in the condition "epithelization of the anterior chamber" following perforating wounds

29. Bullot, G.: J. Physiol. **31**:359, 1904.

30. Fisher, F. P.: Arch. f. Augenh. **102**:146, 1930.

31. Gundersen, T.: Results of Autotransplantation of the Cornea into the Anterior Chamber: Their Significance Regarding Corneal Nutrition, Tr. Am. Ophth. Soc., 1938, to be published.

and especially after operations for cataract. But in this condition the epithelium is not free in the aqueous but is supported by a vascularized framework which probably supplies its nutrition. Again, in epithelial cysts of the anterior chamber the conditions are not analogous to those of my experiment—the cysts contain serum.

It seems likely that the corneal stroma and endothelium can exist on metabolites derived from aqueous alone but unlikely that the epithelium can or does. Its requirements are too high. If the corneal epithelium cannot live in the aqueous humor and if its supply of nourishment from the blood can be almost completely destroyed by peritomy without affecting its existence, then the tears fall under direct suspicion as the nutritive vehicle. The sugar content of tears has not been exhaustively studied, but according to the analyses of Brown [32] it is exceedingly high, 0.65 mg. per hundred cubic centimeters, or approximately six times that of the normal blood sugar.[33]

TABLE 1.—*Nitrogen and Sugar in Aqueous and Tears*

Aqueous (Duke-Elder)	Gm. per 100 Cc.	Tears (Ridley and Brown)	Gm. per 100 Cc.
Total protein nitrogen	0.0236	Total nitrogen	0.158
		Nonprotein nitrogen	0.051
Sugar	0.0983	Sugar	0.650

The following 36 cases show what effect destroying the abnormal blood supply has on the healing of corneal lesions. If direct blood supply were the most important factor in the maintenance of corneal integrity, one certainly would expect that corneal ulcers as well as other inflammatory processes in the cornea would be made worse by destroying this factor. The cases reported comprise a consecutive but carefully selected group of cases of various types of chronic keratitis in which the condition did not yield to customary methods of treatment. On each patient some form of peritomy was done, and the effect of the operation on the corneal lesion was carefully noted. The first patients were treated six years ago, the majority over four years ago and only one within the past year. Thus it has been possible to follow the late results.

The operative procedure was chosen to satisfy the requirements in each case. Neither complete peridectomy (syndectomy), as first suggested by Furnari (1862), involving the excision of a wide band of conjunctiva around the limbus, or Denig's modification, the transplanting of buccal mucous membrane, was employed.

32. Brown, cited by Ridley, F.: Brit. J. Exper. Path. **11**:217, 1930.

33. In a preliminary examination of the sugar content of tears, I have been unable to confirm these findings.

A careful preoperative study with the slit lamp is imperative. Each vascular trunk should be noted and its exact location at the limbus and its depth in the corneal stroma drawn on a schema. Particular attention should be directed toward the location of the arteries, since the obliteration of these is more difficult than that of the large veins. The latter are more obvious and usually more superficially situated in the cornea. If blood vessels are incised but not thoroughly coagulated, corneal hematomas develop quickly. Although they were not found to be injurious, they are disfiguring and absorb slowly. In case 1 (A. R.) and case 2 (J. P.) hematomas were found developing promptly after operation, but their progress was stopped immediately by locating with the slit lamp the large "feeding arteriole" and obliterating it with the diathermy needle.

The combination of diathermic cutting and coagulating current produced by a standard unit served the purpose efficiently, with less trauma and probably with a more discouraging effect on regeneration of the vessels than other cutting instruments. Individual vessels or groups of vessels were severed where they crossed the limbus by a few cutting strokes with a sharp-pointed needle, the point of which was carried as deeply into the stroma as was necessary. In several instances the conjunctiva was dissected from the limbus and the underlying sclera in order to make possible the coagulation of the reticular supply. The importance of the latter has been duly emphasized by Jameson.[34] This procedure was of value when the corneal vessels were at the level of Descemet's membrane. When vessels are at this level, direct attack at the limbus might be hazardous and attended by perforation of the anterior chamber, although this accident has never occurred in my experience. More postoperative reaction occurs in the eye when the reticular supply is disturbed after conjunctival dissection than when a purely local attack is made at the limbus with the diathermy needle. Transitory hypotony was observed in a few cases, corroborating Jameson's observations; transient wrinkling of the posterior corneal surface was observed in others.

Postoperative care was not considered particularly important. Patients were not hospitalized unless an extensive operation was done, since ordinarily but little ocular discomfort followed operation. A monocular bandage was applied under dark glasses for a few days until the wound was covered by new epithelium. Mildly antiseptic collyria were used at intervals of four hours (0.1 per cent zinc sulfate in a

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34. Jameson, P. C.: Vascularization of the Anterior Segment of the Eye: The Bearing of These Studies on Some Operative Procedures, Including the Possible Supplementary Procedure for Glaucoma; Preliminary Report, Arch. Ophth. 9:523 (April) 1933.

saturated solution of boric acid has been found satisfactory), and if secondary iritis existed, an appropriate mydriatic was used.

The following 36 consecutive cases are grouped as follows:

Group 1 consists of 6 cases in which ulcers developed associated with corneal vascularization following herpes corneae—keratitis metaherpetica. In these cases, as in those previously reported by me,[31] herpes virus could not be demonstrated in the ulcers.

Group 2 consists of 7 cases in which the etiologic factor was not clear, but the patients were thought to have ocular rosacea.

Group 3 consists of 12 cases of primary superficial keratitis, probably tuberculous in origin (chronic phlyctenulosis?).

Group 4 is composed of 2 cases in which keratitis followed injuries.

Group 5 consists of 2 cases of progressive yellow stromal infiltration (lipin interstitial keratitis). The etiologic factor in these cases was not clear, although at the onset it was considered tuberculosis.

Group 6 is composed of 5 cases of advanced trachoma.

Group 7 consists of a case of indolent hypopyon ulcer.

Group 8 consists of a case of ulcer of unknown origin, which would not fit into any of the preceding groups.

GROUP 1: DENDRITIC KERATITIS (KERATITIS METAHERPETICA)

CASE 1.—A. R., a man aged 42, was first seen on July 1, 1932, with an initial attack of dendritic keratitis of six days' duration. The cornea stained with fluorescein, as shown in figure 1 *A*. There were no corneal scars. Inoculation of a rabbit gave positive results. Epithelium was removed with an iodized toothpick swab on July 5. Six days later the eye was white and quiet and did not stain. On December 2 there was a minimal recurrence, the ulcer measuring 1 by 1 mm. (fig. 1 *B*). There was no congestion. Inoculations of a rabbit (two attempts) gave negative results. The ulcer healed five weeks after the onset, conservative treatment only being given. On March 14, 1933, there was a second recurrence, the ulcer measuring 1 by 2 mm. There was lacrimation but no congestion. Inoculations of a rabbit (two attempts) gave negative results. Superficial blood vessels were seen in the cornea. No improvement occurred following the usual treatment for ulcers, and a second smaller ulcer developed seven weeks after the onset. Vision was reduced to 20/40. General physical examination gave negative results except for a mild chronic eczema which had been present for ten years. During 1926 the patient had been treated successfully in the outpatient department of the Massachusetts General Hospital for mild prostatitis. He had had no recurrence of this condition. No other focal infection could be found. On May 5, 1933, vascularized ulcers (fig. 1 *C*) developed. By means of the diathermy needle the blood vessels were obliterated at the limbus (fig. 1 *D*). With the slit lamp it was then noticed that there was free blood outside the corneal vessels. There was no venous circulation, but near the limbus along the lower border of the fasciculus of vessels a single arteriole was seen with each pulsation to extravasate blood into the stroma. It became obvious that a large hematoma would develop in the corneal substance. This feeding arteriole was then coagulated, and there was no further escape of blood into

the tissue. Subsequently the patient made daily visits to the outpatient department, where sketches of the eye were made whenever there was any obvious change. The cauterized area was completely reepithelized in five days; there was no corneal staining after eleven days. Red blood corpuscles were present in the hematoma for fifty days. A few blood vessels had budded and crossed the limbus for a distance of 2.5 mm., but not until the ulcer had healed and the hematoma had been practically absorbed.

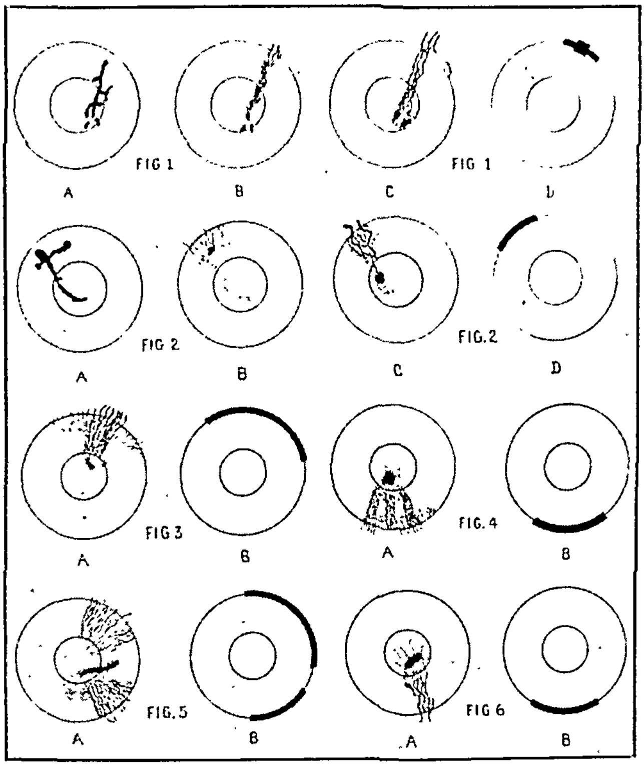
Case 2.—J. P., a woman aged 72, was first seen on March 20, 1933, with an initial attack of dendritic keratitis of six days' duration (fig. 2 *A*). She would not give permission for iodization. She was advised to use antiseptics, mydriatics and fomentations. Vision was 20/70. On March 24 inoculation of a rabbit gave positive results. On April 11 general improvement was noted. The staining area was reduced to a single spot 1 mm. in diameter and there was only faint congestion. Early vascularization occurred (fig. 2 *B*). Examination on April 27 did not reveal any symptomatic improvement. The ulcer was slightly larger and nearer the pupillary space. The vascularization increased. There were many superficial vessels and a few in the midstroma. Inoculation of a rabbit gave negative results. Examination on September 6 showed the ulcer in the upper part of the pupillary space, with capillary loops on its limbal margin (fig. 2 *C*). On September 7 the blood vessels were obliterated at the limbus with a diathermy needle (fig. 2 *D*). The corneal tissue was incised to approximately one-third its depth. Examination with the slit lamp afterward showed complete hemostasis. Examination on September 8 showed that a thin hematoma had developed in the scarred area. Several folds in the corneal endothelium and a few cells in the aqueous were visible. The site of operation reepithelized in five days. The ulcer gradually diminished in size and ceased staining thirty-two days after operation. The visual acuity returned to 20/30. There have been no recurrences. A few small blood vessels have grown into the old scar.

Case 3.—M. McH., a woman aged 32, had her first attack of keratitis, probably dendritic, in November 1933, following pneumonia. There was subsequent blurring of vision. She was first seen on Feb. 7, 1934. The eye had been constantly inflamed for the preceding eight weeks. There was a typical metaherpetic ulcer, 1 by 2 mm., with marked vascularization (fig. 3 *A*). Inoculation of a rabbit gave negative results. Vision was 20/50. On February 8 vascular obliteration with a diathermy needle was carried out (fig. 3 *B*). On February 12 the ulcer no longer stained with fluorescein. There was marked symptomatic improvement. On March 7 there was a recurrence of a single point of stain in the same area. On March 15 the ulcer continued to stain. There was some revascularization of old channels through a deep blood supply which had not been interrupted by the first operation. The deep vessels were obliterated. On March 19 there was no corneal staining. Vision was 20/20. In a follow-up letter in August 1935 the patient stated that there had been no recurrence of symptoms.

Case 4.—A. R., a man aged 24 (a patient of Dr. W. L. Curran), was first seen on March 8, 1935. A foreign body had been removed from the left cornea two years previously followed by an uneventful convalescence. Four months previously a typical dendritic keratitis developed in the left eye, which was treated with tincture of iodine seven days after the onset. After a short period of complete reepithelization of the cornea, keratitis metaherpetica developed, and a small corneal ulcer had been present ever since. Several applications of iodine to the ulcer had failed to effect a cure. The eye showed faint congestion and a

TABLE 2.—*Summary of Cases of Dendritic Keratitis—Keratitis Metaherpetica (Group 1)*

No. of Case	Summary	Result
1	*Ulcer of 7 weeks' duration; arrested in 11 days; no recurrence*	*Excellent*
2	Ulcer of 25 weeks' duration; arrested in 32 days; no recurrence	Good
3	Ulcer of 10 weeks' duration; arrested in 39 days (2 operations); no recurrence	Good
4	Ulcer of 16 weeks' duration; arrested in 4 days; dendritic keratitis 1 year later	Excellent
5	Ulcer of 10 weeks' duration; arrested in 4 days; no recurrence	Excellent
6	Ulcer of 1½ weeks' duration; arrested in 4 days; no recurrence	Excellent



Figs. 1–6.—Diagrams illustrating cases of dendritic keratitis.

staining ulcer, 1 by 2 mm., in the lower part of the pupillary space. According to Dr. Curran's report, the ulcer had migrated from a position near the lower limbus to the position shown in figure 4 *A*. There was superficial scarring in the lower part of the cornea. Many superficial and a few deep blood vessels entered the area. The visual acuity was reduced to 20/40. Obliteration of the pericorneal blood vessels was performed from 4:30 to 7:30 o'clock, local anesthesia being used. The diathermy needle was carried well into the corneal stroma at the limbus in the region of the deep blood vessels (fig. 4 *B*). Examination with the slit lamp after the operation showed complete hemostasis. On March 22 Dr. Curran reported that the eye showed rapid improvement after operation. The ulcer ceased staining in four days, and the eye was practically

white in ten days. On March 15, 1936, the patient was seen again, having been well until two months before, when a second attack of dendritic keratitis developed. This was successfuly treated by the application of iodine.

CASE 5.—M. P. D., a girl aged 6 years, was first seen on Sept. 20, 1934. The child had had three previous attacks of inflammation of the left eye, presumably dendritic keratitis. The present attack was of ten weeks' duration and the symptoms did not abate. The left eye showed slight to moderate congestion, marked infiltration, old scarring and vascularization (chiefly superficial). The ulcer stained (fig. 5 *A*). Inoculation of a rabbit gave negative results. The Wassermann reaction of the blood was negative. On September 22 the superficial vessels at the limbus were obliterated with the diathermy needle (fig. 5 *B*). The ulcer ceased staining four days after operation. The patient remained in the Massachusetts Eye and Ear Infirmary for one week. The eye was white and quiet before discharge and remained so for the following two weeks. The patient has not been seen since.

CASE 6.—H. E. W., a woman aged 71, was first seen on May 7, 1937. The left eye had been moderately sore and red for three days. A previous attack in this eye two years before lasted three months. There was a corneal ulcer 2 by 1 mm. in size in the lower outer part of the pupillary space, with a prominent fasciculus of blood vessels extending to its edge. There were considerable scarring and infiltration surrounding the ulcer and a few folds in the posterior corneal surface but no cells in the aqueous and no keratitis punctata (fig. 6 *A*). Marked corneal hypesthesia was present.

No improvement followed seven days of treatment with a bandage, atropine and mild antiseptics. Obliteration of the superficial pericorneal vessels was performed on May 14 (fig. 6 *B*). On May 18 the ulcer no longer stained, and on May 28 there was no congestion. There has been no recurrence. The orignal attack was probably herpes corneae.

GROUP 2: OCULAR ROSACEA

CASE 7.—M. F. M., a man aged 64, was first seen on May 22, 1934. He gave a history of recurring attacks of inflammation in the right eye for fourteen years. He had been seen in the ophthalmic clinic at intervals for ten years. The present attack was of six months' duration. The eye was moderately congested, and in the lower part of the pupillary space there had developed a nodular, nonstaining corneal lesion surrounded by infiltrates and scars (fig. 7 *A*). There was marked vascularization in all corneal layers, particularly in the deep stroma. There were no cells in the aqueous and no keratitis punctata. The visual acuity was 20/20. There was marked facial rosacea. The intradermal reaction to 0.1 mg. of old tuberculin was negative. On May 23 corneal blood vessels were incised at the limbus with a diathermy needle (fig. 7 *B*). The incision was carried into the depths of the stroma, probably through two thirds of its thickness, and all the vessels except those on Descemet's membrane were interrupted. Little reaction followed the operation, but a small hematoma developed in the cornea, with some keratitis striata, which extended upward into the pupillary space. On June 8 the patient was asymptomatic, and the eye showed no congestion. On July 2 a slight recurrence of symptoms was noted, which had had its onset twelve days previously, with the formation of a new small corneal infiltrate near the limbus at 8 o'clock. By July 7 the recurrence had subsided completely. The report of Dec. 10, 1935, stated that the patient had been practically well since his last attack. The eye

was occasionally slightly irritable, but attacks were of only one or two days' duration. The cornea showed no new scars. The visual acuity was 20/20.

Case 8.—M. R., a woman aged 72, was first seen on April 29, 1935. Both eyes had been constantly irritated for six years. The right eye showed slight congestion. There were marked keratitis epithelialis disseminata, with thickening and desquamation of epithelium, most marked in the lower part of the cornea, and much punctate staining. Superficial vascularization of the cornea (fig. 8 *A*), slight marginal atrophy of the iris and moderate retinal arteriosclerosis were present. There was diminished lacrimation in each eye (a 5 mm. filter paper moistened 4 mm. in five minutes). The pericorneal blood vessels were normal. There was moderate facial rosacea. The visual acuity was 20/70. On July 18 the pericorneal blood vessels were obliterated with a diathermy needle from 2 to 10 o'clock (fig. 8 *B*). On July 22 the wound showed no staining. The entire cornea was smooth and did not stain except in the quadrant of the cornea adjacent to the untreated area. There was some subjective improvement. On August 5 no change was noted. On September 25 there was a slight recurrence of staining over the entire cornea. The visual acuity was 20/40.

Case 9.—E. F. S., a man aged 41 (a patient of Dr. W. B. Lancaster), had his first attack of ocular inflammation in August 1933 during a long motor trip, at which time the eyes were exposed to much wind and glare. Bilateral corneal ulcers developed, which persisted for five weeks. A similar attack, lasting ten days, occurred during May 1934, and a third attack, lasting seven days, during November 1934. The fourth attack, the only one observed by me, began Jan. 31, 1935. It was by far the most severe. Both eyes showed extreme photophobia and marked congestion. There were multiple discrete gray infiltrates in the epithelium and anterior stroma of each cornea. Less than half of the lesions stained. There was superficial vascularization from many points about the limbus. No iritis was present. The lids were inflamed but showed no evidence of trachoma. Conjunctival smears showed no eosinophils. There were a few staphylococci and Bacillus xerosis in smears as well as in cultures. Physical examination gave essentially negative results, except for facial rosacea. The intradermal reaction to 0.1 mg. of old tuberculin was strongly positive. Roentgen examination of the chest gave negative results. There was evidence of a slight nonspecific chronic prostatitis. Gastric analysis showed a normal hydrochloric acid content. The patient showed no improvement after treatment at home for eighteen days and was admitted to the Massachusetts Eye and Ear Infirmary on February 17. The right cornea was more involved than the left and showed a large area of infiltration in the lower part of the pupillary space. There were smaller infiltrates (fig. 9 *A*) in the upper half of the cornea, with superficial vascularization. On February 19 the conjunctiva was loosened from the limbus above, and the blood vessels crossing the corneal limbus were obliterated (fig. 9 *B*). The patient remained in the hospital for twenty-one days, during which time there was slight though gradual improvement in each eye. He was then transferred to a convalescent hospital, where he remained for twelve weeks. He has since returned to his work as a practicing dentist and has had no subsequent attacks.

Case 10.—A. DeS., a woman aged 29, had suffered from inflammation of the right eye for the first time six years before the present attack. She had received treatment at another hospital and in the outpatient department of the Massachusetts Eye and Ear Infirmary, and there had been some regression of symptoms at

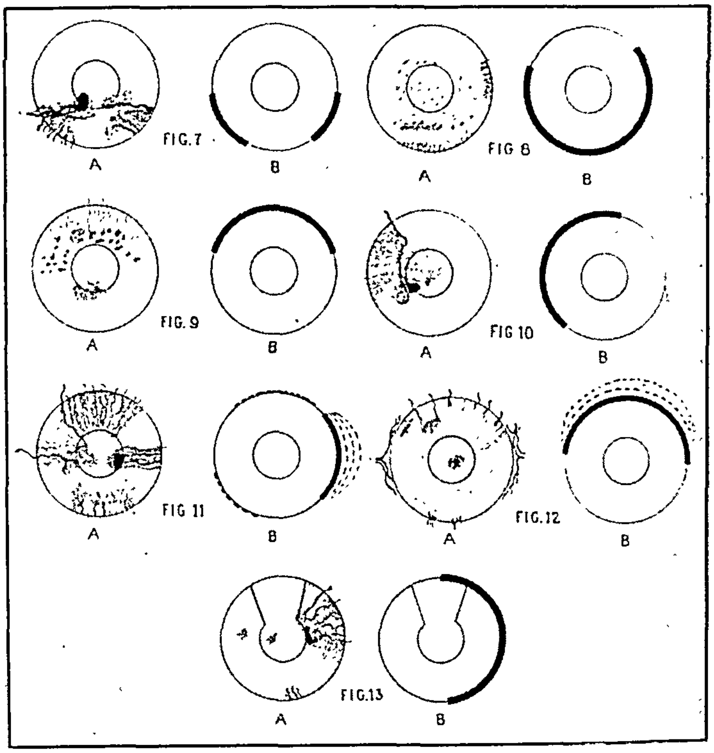
times, but many periods of acute exacerbation, and no definite improvement from local treatment. There was moderate facial rosacea. The Wassermann reaction of the blood was negative. The intradermal reactions to 0.01 mg. of old tuberculin were negative. Little hypesthesia was present. There was a fairly dense superficial infiltrate in the upper outer corneal quadrant (fig. 10 *A*). Superficial vascularization was marked, and there was some deep vascularization throughout the lesion. There was an ulcer, 1.5 by 2 mm., at the end of the fasciculus. On Sept. 4, 1935, the superficial vessels at the limbus were obliterated (fig. 10 *B*). Two days after operation the eye felt much better, and there was a definite diminution in the size of the ulcer. The staining area measured 1 mm. in diameter. By September 9 the eye was almost quiet, and the ulcer was reduced to the size of a pinpoint. The vessels showed some return of circulating blood. On October 18 it was found that there had been a recurrence of symptoms, and the ulcer had increased to its original size. The blood vessels were again obliterated in the same area. Again a definite shrinking in the size of the ulcer was observed, and three weeks after operation the eye was quiet and the cornea did not stain.

Case 11.—C. M., a woman aged 48 (a patient of Dr. J. J. Regan), had had continuous inflammation of the right eye since 1930, except for a complete remission of twelve months during 1934 to 1935. Tuberculin therapy was administered during 1933, 1934 and 1935. During July 1935 the eye again became inflamed, and the inflammation continued unabated until November 26, when the patient was first seen by me. The right eye was moderately congested and showed multiple areas of scarring and infiltration (fig. 11 *A*). There was marked superficial and deep vascularization, with an ulcer 2 mm. in diameter in the nasal border of the pupillary space. No posterior corneal deposits could be seen. The aqueous was too obscured for careful study. The visual acuity was 4/200. The left eye showed a superficial stromal scar 2 mm. in diameter in the upper outer corneal quadrant. General physical examination gave negative results except for moderate facial rosacea. No focal infections were found. On November 26 the conjunctiva was dissected from the limbus and underlying sclera, from 2 to 4 o'clock, and the superficial blood vessels at the limbus and on the sclera were obliterated with the diathermy needle. Likewise the individual blood vessels were obliterated at the limbus in the upper and lower temporal quadrants (fig. 11 *B*). The retracted conjunctiva was then pulled toward the cornea and sutured in position. Examination on December 2 showed the eye to still be moderately congested, but the ulcer showed no staining, although it was still slightly faceted. The eye became entirely white during the succeeding month, and the patient was asymptomatic when last seen on March 5, 1936.

Case 12.—E. R., a man aged 50, experienced his first attack of ocular inflammation during 1926. Since then a great many corneal ulcers had appeared on both eyes. The visual acuity during the six months preceding the patient's admission to the hospital had diminished, so that he had been forced to abandon an active surgical practice. No treatment had given any permanent relief. Careful physical examination revealed only the following abnormalities: marked facial rosacea, a basal metabolic rate of —10, mild achylia gastrica and apical scars in both lungs. Examination on Nov. 19, 1935, showed slight congestion of the right eye and moderate congestion of the left eye. There were diffuse infiltration and superficial scarring of both corneas. The epithelium was thickened and slightly rough. There was slight superficial vascularization, especially in the upper part of the left cornea from 10 to 2 o'clock, extending 3 mm. beyond the limbus. There was a

TABLE 3.—*Summary of Cases of Ocular Rosacea (Group 2)*

No. of Case	Summary	Result
7	Localized keratitis, 27 weeks' duration; arrested in 16 days; slight temporary recurrence 6 weeks after operation	Excellent
8	Marked keratitis epithelialis, probably of several years' duration; striking immediate improvement; no permanent cure	Fair
9	Severe keratitis, 7 weeks' duration before operation; gradual improvement not directly related to operation	No improvement
10	Severe recurring ulcers of 6 years' duration; marked reduction in size of ulcer after each of 2 operations; no cure	Fair
11	Ulcer of 20 weeks' duration; arrested in 6 days; no recurrence	Excellent
12	Small ulcer in pupillary space, of unknown duration (probably many weeks); arrested in 2 days; no improvement in general condition	Fair
13	Severe ulcer of 16 days' duration; arrested in 4 days; no recurrence	Excellent



Figs. 7–13.—Diagrams illustrating cases of ocular rosacea.

staining ulcer 1 mm. in diameter just below the corneal center (fig. 12*A*). No deposits were seen on the posterior corneal surface, nor were any cells observed in the aqueous. There was no corneal hypesthesia. Epiphora was marked in both eyes. On November 20 obliteration of the pericorneal blood vessels was performed. The conjunctiva of the left eye was loosened from the limbus from above, from 9 to 3 o'clock, and the anterior ciliary vessels were lightly coagulated with the diathermy needle for a distance of 3 mm. behind the limbus (fig. 12*B*). The patient made an uneventful convalescence and was discharged from the hospital on November 28. The ulcer showed no staining twenty-four hours after operation, but considerable keratitis epithelialis remained in the lower half of the cornea. After the operation there developed in the posterior corneal surface many fine folds, which persisted for one week. The eye remained rather irritable and

teared profusely, but showed gradual improvement up to the time of the patient's discharge from the hospital on November 28. He was given general and local treatment for the ocular rosacea. Since discharge there has been some improvement in the ocular rosacea but there have been recurrences of the keratitis.

Case 13.—J. K., a man aged 36, was first seen on March 9, 1936. The left eye had been sore and red for seven days. Five years previously the patient had been under treatment for six months in the Massachusetts Eye and Ear Infirmary for a severe attack of keratitis, secondary iritis and glaucoma. Several paracenteses and iridectomy were performed. The cause of the keratitis could not be determined, although it was thought to be the type which is associated with acne rosacea. The eye showed marked congestion, and on the cornea there was a highly vascularized ulcer, 2 by 3 mm. in size, 4 mm. from the limbus in the upper outer quadrant. There were several isolated corneal scars (fig. 13 *A*). Slight secondary iritis was present, but the intraocular pressure was not elevated. The eye was treated with atropine, zinc oxide and an ichthammol ointment and was bandaged, but no improvement occurred. On March 18 the superficial pericorneal blood vessels were obliterated from 12 to 6 o'clock (fig. 13 *B*). The eye improved rapidly thereafter. The discomfort ceased twenty-four hours after operation, and four days following there was no staining of the ulcer or operative wound. On March 1 the eye was entirely quiet.

GROUP 3: PRIMARY SUPERFICIAL KERATITIS, PROBABLY TUBERCULOUS

Case 14.—M. C., a woman aged 29, was first seen on March 24, 1933, with an atypical superficial punctate keratitis of both eyes, more active in the right. She complained of recurring attacks of ocular inflammation for the preceding year. Physical examination revealed a tall, thin, frail-looking woman in fairly good health. Roentgen examination of the chest showed old scars in the apexes of both lungs but no active tuberculosis. The reaction to 0.0025 mg. of old tuberculin was positive. The Wassermann reaction of the blood was negative. No focal infections were found. The patient was seen at regular intervals during the following year, and all local and general therapy failed to arrest the inflammation in the right eye. An ulcer, 3.5 by 1 mm., developed in the lower part of the cornea; it became vascularized and gradually advanced into the upper part of the pupillary space (fig. 14 *A*). The visual acuity of the right eye decreased to 20/70 + 2; that of the left eye remained 20/20. There was no evidence of intraocular inflammation, but the eye was constantly congested. On May 23, 1934, the vascularization was chiefly superficial, but there were some stromal vessels also. On May 24 obliteration of the pericorneal blood vessels from 5:30 to 7 o'clock was performed with the diathermy needle, which was carried into the posterior corneal stroma (fig. 14 *B*). Examination on June 8 showed that the ulcer had decreased rapidly in size and ceased staining five days after operation. A small hematoma formed in the cornea, which absorbed completely in ten days. The eye was white and quiet, and the administration of atropine was discontinued. On August 17 it was found that the right eye had remained practically asymptomatic until twenty-four hours previously, when it again became inflamed; it showed slight congestion, and an ulcer 2 mm. in diameter was seen in the lateral portion of the pupillary space. There had been considerable reformation of blood vessels in the previously vascularized area. On August 18 the vessels which had reformed around the margin of the previous wound of operation were obliterated, the same method being used

that was employed previously. The ulcer again showed a rapid diminution in size after operation, but a faintly staining point remained until October 20, when it disappeared and the eye became asymptomatic. On July 25, 1935, it was found that the patient had had no major corneal inflammations since the last operation. On two occasions, minute staining ulcers formed in the previously affected area but healed within a few days.

Case 15.—L. G., a Negro aged 55, first seen on Jan. 11, 1934, had had attacks of painful inflammation in both eyes in childhood. The left eye had always been more involved than the right. The present attack in the left eye was of about ten days' duration. Examination on March 31 showed that the patient had not responded to the regular treatment for ulcer in the ophthalmic clinic. Vision was more blurred (20/40), and there was slight congestion. An ulcer, 1 by 2 mm., was observed in the pupillary area. There was marked superficial and deep vascularization (fig. 15 *A*). Scattered fine deposits were present on the posterior corneal surface. There were few aqueous cells. The Hinton reaction of the blood was negative. The intradermal reaction to 0.1 mg. of old tuberculin was positive. No focal infection was found. Obliteration of the corneal vessels at the limbus from 5 to 7 o'clock was done with a diathermy needle (fig. 15 *B*). The corneal stroma was incised to approximately half of its depth. The majority of the vessels were interrupted. On April 2 there was marked subjective improvement. The ulcer was reduced to one third of its size before operation. On April 7 the eye was white, and the ulcer showed a faint point of stain. On April 11 there was no staining of the ulcer. On June 29 examination revealed that the patient had been practically asymptomatic since the operation. The eye was white and quiet, and there was no staining of the cornea.

Case 16.—M. B., a woman aged 42, first had an ocular inflammation following measles in childhood. Mild attacks occurred again at the age of 34, but no severe inflammation was present until four years before the present attack, when both eyes were inflamed for five weeks. The present attack began in the right eye four months before admission to the hospital and in the left eye two months before. The patient had been under treatment in the outpatient department for a month. Examination showed the right eye to be white and quiet, with a visual acuity of 20/50. There were several punctate and fusiform scars in the superficial corneal stroma, with practically no vascularization. The left eye was slightly congested and showed marked epiphora and photophobia. There was no corneal hypesthesia. There were a diffuse infiltrate in the epithelium and superficial corneal stroma and four discrete staining ulcers (fig. 16 *A*). There was marked superficial vascularization of the cornea extending into the pupillary space, especially in the upper, outer and lower portions. The visual acuity, with a 2 mm. pinhole, was 20/70 + 2. On April 16, 1934, obliteration of the pericorneal blood vessels was performed in the lower half of the cornea with the diathermy needle (fig. 16 *B*). The incision was carried through the epithelium and into the most superficial fibers of the corneal stroma. By April 18 there had been little change in symptoms. There was no staining of the cornea. The globe was still congested. On May 3 the eye was practically white although there was still some photophobia. On Sept. 26, 1935, it was found that both eyes had been asymptomatic, white and quiet since the last visit. The patient felt that the operation had been of definite help. In conjunction with the operative procedure, the patient received a course of injections of tuberculin from March 1, 1934, to Nov. 7, 1935.

Case 17.—In R. C., a Negro girl aged 13, phlyctenular keratitis developed in both eyes at the age of 2 and persisted almost without interruption until the age of 8. There had been a recurrence in the left eye one year before the present attack. Physical examination gave negative results. Roentgen examination of the chest showed a supracardiac shadow, suggesting enlargement of the hilar glands. The intradermal reactions to 0.001 mg. of old tuberculin were positive. The Wassermann reaction of the blood was negative. No form of local therapy had been of any marked benefit. The right eye was white but showed several old corneal scars. The left eye was moderately congested and showed marked photophobia and lacrimation. There was diffuse scarring of practically the entire cornea with many areas of denser infiltration. Scattered irregularly over the cornea were ten staining ulcers, approximately 1 mm. each in size. There was superficial vascularization, most marked from 4 to 6 o'clock (fig. 17 *A*). Examination with the slit lamp showed no evidence of intraocular inflammation. On Jan. 15, 1935, obliteration of the pericorneal blood vessels from 4 to 6 o'clock was carried out with the diathermy needle. The incision was carried into the superficial corneal stroma (fig. 17 *B*). The report of January 19 stated that the patient had had an uneventful convalescence. The symptoms of irritation had largely decreased, and the cornea showed no staining. On Jan. 25, 1936, there was a slight recurrence of symptoms. There were many fine staining points in the lower half of the cornea. On January 30 both eyes were white and quiet. The patient was able to return to school. After operation the patient made several visits to the hospital. On only two visits was there the slightest evidence of corneal inflammation. Examination on March 23 showed that there had been a reestablishment of circulation in a few vessels in the lower portion of the left cornea. The visual acuity was 20/50 in the right eye and 20/30 in the left eye.

Case 18.—P. V., a girl aged 12 years, was seen on July 25, 1935. For the preceding five years, almost continually, she had had a diffuse phlyctenular-like keratitis of both eyes, the cause of which could not be determined. Tuberculin tests had been consistently negative. Physical examination showed no evidence of tuberculosis. Roentgen examination of the lungs showed some increased density of the shadow of each hilus. The Wassermann reaction of the blood was repeatedly negative. No focal infections could be found. Sensitization tests showed no reaction to the usual proteins. The patient was admitted to the Lakeville State Sanatorium on April 12, 1932, and remained there until May 29, 1932. It was then thought that there was insufficient evidence for a diagnosis of tuberculosis to warrant the continuation of care in the sanatorium. Examination on July 25, 1935, showed slight congestion of both eyes, with diffuse scarring of both corneas and many localized areas of denser infiltration. The entire surface of both corneas showed marked vascularization and roughness, and there was some fine punctate staining—keratitis epithelialis disseminata (fig. 18 *A*). Examination with the slit lamp showed no evidence of intraocular inflammation. The visual acuity in the right eye was 20/20 and in the left eye 4/200. Obliteration of the pericorneal blood vessels was performed in the left eye with the diathermy needle (fig. 18 *B*). The incisions were superficial and interrupted, particular attention being directed toward the large blood vessels. On August 7 the eye was white and quiet. The patient had had an uneventful convalescence. Examination on October 2 showed that the left eye had continued to improve. The visual acuity was 8/200.

Case 19.—D. M., a girl aged 7 years, was seen on July 28, 1934. For the preceding five years she had had recurring attacks of phlyctenular keratitis in

both eyes, more frequently in the right. Attacks were often associated with common colds and appeared practically every month except during the summer. General physical examination gave negative results. Roentgen examination of the lungs showed no evidence of tuberculosis. Intradermal reactions to 0.01 mg. of old tuberculin were negative. The right eye was moderately congested and showed several active lesions typical of phlyctenular keratitis. These were concentrated in the upper half of the cornea, where there was marked vascularization downward to the upper pupillary border (fig. 19 *A*). Slit lamp examination could not be made. The left eye showed a similar picture, but much less marked. With the patient under ether anesthesia, obliteration of the pericorneal blood vessels was performed at the upper limbus, from 10 to 2 o'clock (fig. 19 *B*), with the diathermy needle. The patient made a slow but uneventful recovery from the operation. Both eyes have been much improved since operation. The attacks of inflammation have been fewer in number and of less severity. Vascularization of the upper part of the right cornea has returned to a considerable extent, but is less marked than prior to operation.

Case 20.—T. K., a man aged 36, was seen on Oct. 18, 1935, because of moderate to severe symptoms of corneal inflammation of the left eye for three months. There had been a similar attack ten years before, which had lasted for several months. The eye was slightly congested. There were marked lacrimation and photophobia. The cornea was diffusely and irregularly scarred, and there were two staining ulcers (fig. 20 *A*). The corneal epithelium was slightly vascularized at 8 and 11 o'clock, and there were a few vessels in the stroma. No form of local treatment had been of any value. General physical examination gave negative results. The Wassermann reaction of the blood was negative. A few teeth with apical abscesses had been extracted during the preceding attack without benefit. The intradermal reaction to 0.0025 mg. of old tuberculin was positive. There was no corneal hypesthesia. Examination with the slit lamp showed no evidence of intraocular inflammation. The visual acuity was 20/200. Obliteration of the pericorneal blood vessels was performed at the limbus from 6 to 8 o'clock and from 10 to 12 o'clock. The diathermy needle was carried only through the epithelium in these regions (fig. 20 *B*). The eye showed increased redness and was rather uncomfortable for twenty-four hours after the operation, when, on October 28, some improvement was noted. Seven days after operation, although there was slight congestion, there was absolutely no corneal staining. Examination on December 16 showed that the improvement was of short duration. Six weeks after operation there was a small ulcer in the same region and a recurrence of symptoms.

Case 21.—C. P., a man aged 22, was seen on Sept. 25, 1935. At the age of 6 years, he had his first attack of keratitis in the right eye which was of a few weeks' duration. Two years before the present attack he had a recurrence, lasting six weeks. A third attack, lasting only a week, developed nine months before. After this attack the eye was well for two weeks, then again became inflamed. The eye was particularly sensitive during warm weather. Photophobia and epiphora were predominating symptoms, but there was not much pain. The eye was slightly congested. The cornea showed diffuse superficial scarring with mottling due to areas of increased density. The corneal epithelium was slightly rough throughout. A staining ulcer, 1 by 2 mm. in diameter, was present (fig. 21 *A*), and there was practically uniform superficial vascularization about the periphery of the cornea, extending over the limbus from 3 to 4 mm. Examination with the slit lamp showed no evidence of intraocular inflammation. General

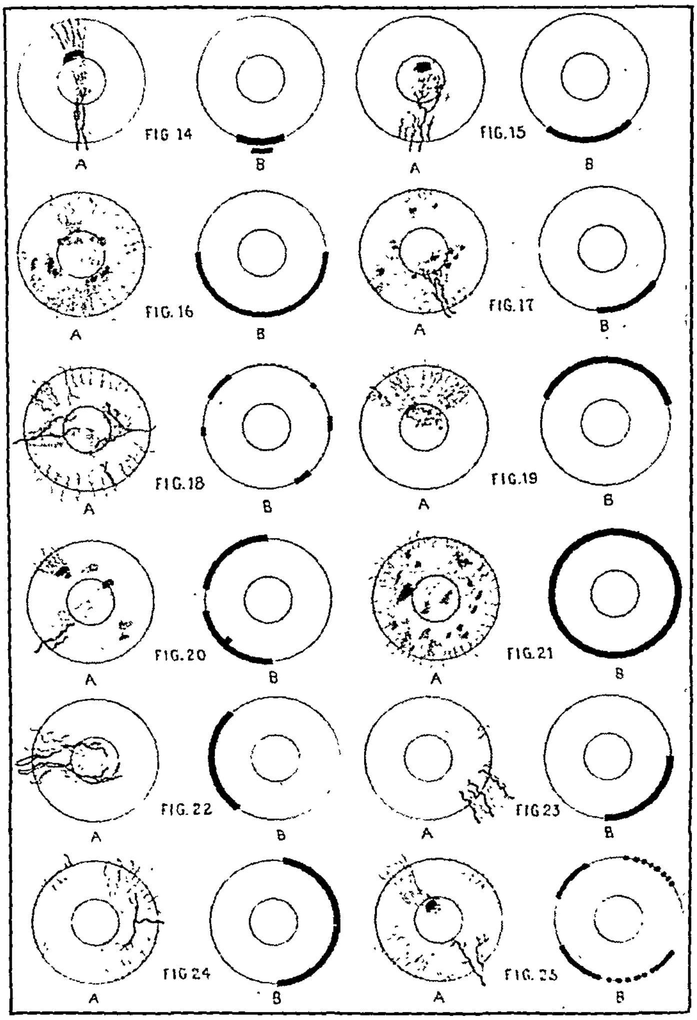
physical examination gave negative results. The Wassermann reaction of the blood was negative. Intradermal reactions to 0.01 mg. of old tuberculin were negative. Roentgen examination showed normal accessory nasal sinuses and an apical abscess of one tooth, which had been treated. The visual acuity was reduced to 20/40. Complete obliteration of the pericorneal blood vessels was done with the diathermy needle; the point was carried through the epithelium of the limbus (fig. 21 *B*). The patient had some discomfort for two hours after operation. On September 28 the eye showed moderate congestion but no staining of the ulcer or of the wound. On October 14 the right eye was completely white and quiet, and the cornea showed no staining. On October 21 the patient again had some symptoms of irritation in both eyes. There was a staining area the size of a pinpoint in the upper part of the right cornea. The visual acuity had returned to 20/20. The patient has not returned for observation since October 21 and presumably is well.

Case 22.—L. B., a girl aged 4 years, was seen on Feb. 21, 1935. She was referred from the Blind Babies' Nursery, where she had been treated for recurring attacks of phlyctenular keratitis of both eyes since infancy. The left eye had been inflamed and had resisted treatment for three months prior to the present examination. There was marked irregularity of the corneal surface, with dense infiltration and superficial vascularization (fig. 22 *A*) and some folliculosis in both fornices. Obliteration of the pericorneal blood vessels was performed at the limbus from 7 to 10 o'clock with the diathermy needle (fig. 22 *B*), local anesthesia being used. The patient was immediately returned to the institution. On December 10 a report by Dr. H. B. C. Riemer, oculist of the Blind Babies' Nursery, stated: "The eye was white and quiet seven days after operation. There has been considerable improvement in the amount of inflammation present. The patient has had two minor attacks of inflammation since, each lasting three or four days."

Case 23.—R. E. K., a man aged 48 (a patient of Dr. M. J. King) was seen on April 3, 1934. The right eye first became sore ten months before. He had had recurring attacks of inflammation since then, with remissions lasting no longer than from seven to ten days. During these attacks the eye became congested and slightly sore and showed considerable photophobia and lacrimation. There were a moderately dense scar in the upper inner quadrant of the cornea and a large, broad, well vascularized pseudopterygium crossing the limbus for a distance of 2.5 mm. in the lower inner quadrant (fig. 23 *A*). At times there had been small staining ulcers at the border of the pseudopterygium. Examination with the slit lamp showed no evidence of intraocular inflammation. General physical examination gave negative results. The Wassermann reaction of the blood was negative. No focal infections were found. The intradermal reaction to 0.0025 mg. of old tuberculin was positive. The patient had received treatment with tuberculin for six months before the present examination. Obliteration of the pericorneal blood vessels from 3 to 6 o'clock was performed with the diathermy needle. The incision was carried into the superficial corneal stroma at the limbus (fig. 23 *B*). According to a report on April 10, the patient had considerable pain for twenty-four hours after operation, and the cornea showed staining of the wound for forty-eight hours, with a slight hematoma in the region of the pseudopterygium. Marked improvement occurred during the four days prior to April 10. A report on June 16, 1936, showed that the patient had continued to improve and that there had been practically no attacks since operation.

TABLE 4.—*Summary of Cases of Primary Superficial Keratitis, Probably Tuberculous (Group 3)*

No. of Case	Summary	Result
14	Ulcer of over 12 months' duration; arrested in 9 days; recurrence in 3 months	Fair
15	Ulcer of 13 weeks' duration; arrested in 11 days; no recurrence	Excellent
16	Multiple small ulcers of 9 weeks' duration; arrested in 6 days; also treated with tuberculin	Fair
17	Multiple fine ulcers of lower part of cornea of 12 months' duration; arrested in 9 days; minimal recurrence; marked general improvement	Good
18	Severe superficial keratitis with punctate staining of several years' duration; eye white and quiet in 3 weeks	Good
19	Severe superficial keratitis of several years' duration; no definite improvement	No improvement
20	Ulcer of 3 months' duration; much improved 7 days after operation; recurrence in 6 weeks	No improvement
21	Ulcer of 8 months' duration; completely arrested in 14 days; slight recurrence 2 weeks later	Fair
22	Severe, superficial keratitis, 3 months' duration; arrested in 7 days; slight recurrence	Fair
23	Regularly recurring ulcers at head of pseudopterygium for 8 months; healing of ulcers in 4 days; marked reduction in recurrences; tuberculin therapy also	Fair
24	Pseudoptergyium for 20 years; occasional attacks of inflammation; ? of diminution in number of attacks; tuberculin therapy also	No improvement
25	Pseudopterygium with ulcer at its head, 2 weeks' duration; ulcer arrested in 14 days after transplantation of pseudopterygium and obliteration of limbal vessels; no recurrence	Excellent



Figs. 14–25.—Diagrams illustrating cases of primary superficial keratitis, probably tuberculous.

CASE 24.—T. E. H., a man aged 68 was seen on March 22, 1934. For approximately twenty years he had had recurring attacks of ocular inflammation. Both eyes had large, rather dense pseudopterygia at the inner and outer limbus, more marked at the former. Only the right eye showed active inflammation, and this was slight. The extent of the pseudopterygium and approximate vascularization of the right eye are shown in figure 24 *A*. The vascularization was chiefly superficial, although there were some deep vessels. Examination with the slit lamp showed no evidence of intraocular inflammation. General physical examination showed no systemic disease. The intradermal reaction to 0.0025 mg. of old tuberculin was positive. Obliteration of the pericorneal blood vessels from 12 to 6 o'clock was performed with the diathermy needle. The incision was carried into the superficial corneal stroma at the limbus (fig. 24 *B*). On March 24 the eye was slightly congested. There was no staining of the wound. On Jan. 23, 1935, the patient stated that he felt that the right eye had improved considerably since operation. There had been less discomfort, and the visual acuity was slightly better. There has been no advancement in the pseudopterygium since operation. The blood supply has been definitely decreased by the operation and is now considerably less than in the pseudopterygium of the opposite eye.

CASE 25.—A. T. B., a woman aged 46, was seen on Nov. 15, 1935. Five years before she had been treated for four months at a local hospital for a corneal ulcer of the left eye. Three years before a second attack occurred lasting two and a half months. Two weeks before the left eye again became sore and became steadily more inflamed in spite of treatment. The right eye was white and quiet but showed an old scar in the superficial corneal stroma in the upper outer quadrant. The left eye was irritated and showed moderate congestion in focal areas surrounding the limbus. There was marked roughness of the corneal surface and about the periphery were patchy areas of infiltration which were well vascularized to the extent of a pseudopterygium formation at 7:30 and 10:30 o'clock. At the end of the upper pseudopterygium there was a staining ulcer, 1 by 2 mm. (fig. 25 *A*). There were a few cells in the aqueous. The visual acuity was 20/50. General physical examination gave negative results except for moderate facial rosacea. The intradermal reaction to 0.0025 mg. of old tuberculin was negative. In spite of the negative test for tuberculosis and the presence of facial rosacea, the corneal lesions appeared to be most characteristic of tuberculosis. The upper pseudopterygium was dissected from the cornea and transplanted under the conjunctiva above, local anesthesia being used. The lower pseudopterygium was excised, and the cut blood vessels were coagulated by the diathermy needle. Also, the individual vessels which could be seen were cut off at the limbus with the diathermy needle (fig. 25 *B*). According to the report of November 29 there was considerable added discomfort for two days after operation. Congestion increased considerably, and several scattered hematomas developed under the epithelium. There was then marked improvement in symptoms and the corneal ulcer healed on the fourteenth postoperative day. Examination on December 18 showed no recurrence. The eye was completely white and quiet.

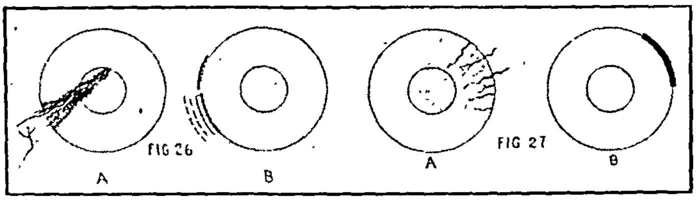
GROUP 4: KERATITIS FOLLOWING TRAUMA

CASE 26.—A. F., a boy aged 14 years, on Aug. 11, 1928, had sustained a nonperforating laceration of the right cornea from the limbus at 8 o'clock to the upper pupillary area and was successfully treated in the outpatient department. He was first seen by me on July 1, 1931. The patient thought that the scar was growing.

It was vascularized throughout. The eye was white, and the visual acuity was 20/20. On Jan. 31, 1933, there was slight congestion of the scar and adjacent limbus. On December 19 the patient complained that the eye became sore and congested at intervals, lasting a few days; the eye was particularly sore when the patient had an infection of the upper part of the respiratory tract. The scar was prominent and markedly vascularized from the midstroma outward. There were from five to ten staining dots along the lower border of the scar in the pupillary space. There were no cells in the aqueous. The visual acuity was 20/30. According to the report of Feb. 23, 1934, the eye became very sore twenty-four hours previously after vigorous physical exercise and exposure to cold. The patient had

TABLE 5.—*Summary of Cases of Keratitis Following Trauma (Group 5)*

No. of Case	Summary	Result
26	Recurring ulcers in old corneal laceration, 10 weeks' duration; arrested in 7 days after vascular obliteration	Excellent
27	Keratitis resulting from mustard gas; tuberculous keratitis (?), 17 years' duration; deep vessels incised with keratome	No improvement



Figs. 26–27.—Diagrams illustrating cases of keratitis following trauma.

a "cold." The eye was slightly congested, and there was no pus in the conjunctival sac. There was a staining linear ulcer along the lower border of the scar. The entire lesion was red and slightly elevated (fig. 26 *A*). There were many cells and some fibrin in the aqueous. The visual acuity was 20/100. On February 26, while rubbing the eye, the patient experienced a sudden pain, and there was a gush of fluid from the eye. Examination the next day showed a perforated ulcer with a prolapsed iris. The anterior chamber reformed. The patient was admitted to the infirmary for immediate treatment. Operation was performed by Dr. F. H. Verhoeff. There was a single corneal suture over the ulcer. Attempts to reduce the incarcerated iris through the wound failed. A small keratome incision was made temporarily above the fasciculus. The anterior chamber refilled with saline solution. The iris was dislodged from the ulcer with a spatula. The suture was tied. The conjunctiva and vessels entering the scar were incised, and the vascular reticulum was scarified (fig. 26 *B*). On March 7 the patient was discharged from the infirmary. There was slight congestion and the iris was free from the cornea. There was no corneal staining, and few cells were seen in the aqueous. On March 22, the eye was white. By October 26 there had been no return of symptoms since operation. The visual acuity was 20/15.

CASE 27.—J. S., a man aged 36, was seen on Feb. 21, 1935. He had received a mustard gas burn of both corneas while serving in the American Expeditionary Forces in 1918. Both eyes had been inflamed almost constantly ever since, totally incapacitating the patient. He had had thorough ocular and physical examinations

at the Walter Reid Hospital. No definite diagnosis had been made. He had been under continuous treatment at the Massachusetts Eye and Ear Infirmary since 1931, with practically no improvement. Focal infections had been eliminated. Repeated physical examinations gave completely negative results. The intradermal reaction to 0.0025 mg. of old tuberculin was positive.

The right eye was more involved than the left. It showed a dense infiltration of the cornea, occupying practically the entire nasal half. The corneal surface was irregular. There were many deep and a few superficial vessels entering this area between 1 and 4 o'clock (fig. 27 *A*). The eye had shown a little evidence of intraocular inflammation, but only occasionally. There was slight congestion, but marked irritation. A keratome incision was made at the limbus from 1:30 to 9 o'clock in an attempt to sever the blood supply of this area of inflammation (fig. 27 *B*). On March 28 there were no untoward consequences from the operation. Slight hyphemia developed, but this disappeared within a week. The eye whitened to its previous condition within two weeks and has shown no change since. In the deep stroma of the cornea there was some postoperative hemorrhage, but this also disappeared. Examination on June 11, 1936, showed that the eye continued to be irritable. There was no benefit from the operation.

GROUP 5: LIPIN INTERSTITIAL KERATITIS

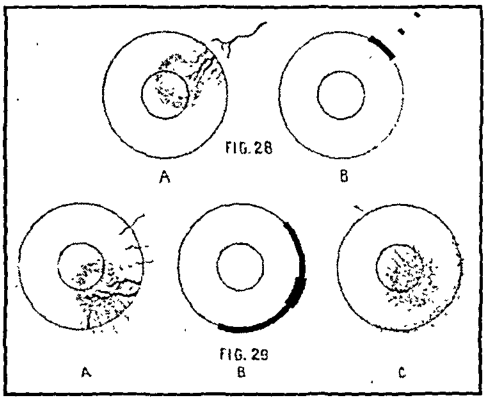
Case 28.—K. D., a woman aged 47, was seen on April 16, 1934, complaining of slight irritation of the left eye. Examination showed faint congestion, and there was a fresh area of infiltration in the central and upper portions of the cornea, evidenced by edema of the corneal surface, posterior bowing and many folds in the posterior surface. There were a few cells in the aqueous and scattered deposits on the endothelium. The intraocular pressure was slightly reduced. The visual acuity was 15/200. This condition of the cornea remained almost stationary for the following six months, when, in spite of diminishing congestion, there was an increase in the amount of infiltration in the posterior corneal stroma, consisting of a deposition of fine white spiculated, crystalline-looking material. There had been some ingrowth of blood vessels along Descemet's membrane, although there was some diminution in the number of folds of the posterior surface and in the amount of edema of the anterior surface. There was a gradual increase in the white deposit in the anterior corneal stroma. The deposit assumed a discoid shape, with slight clearing toward the center. There was also in this area a considerable amount of amorphous yellowish deposit, which appeared to be lipoidal. Sixteen months after the onset the appearance was as shown in figure 28 *A*. General physical examination revealed a rather asthenic-looking woman who was suspected of having tuberculosis, though this was never proved roentgenographically. There were no focal infections. The Wassermann reaction of the blood was negative. The intradermal reaction to 0.005 mg. of old tuberculin was positive. On Aug. 16, 1935, the deep blood vessels were obliterated from 1:30 to 2:30 o'clock as they crossed the limbus (fig. 28 *B*), local anesthesia being used. The diathermy needle was carried into the depths of the stroma without perforating the anterior chamber. There was little reaction after the operation. On August 27 examination with the slit lamp showed complete obliteration of practically all the deep stromal vessels. There was still some faceting of the operative wound. As far as could be determined from photographs taken before operation, there had been no advance in the amount of corneal infiltration up to Feb. 15, 1936. There had been some reestablishment of circulation to the area, but only a small fraction of what it was before operation.

Case 29.—G. K., a man aged 52, had been under constant observation and treatment for six years for an obstinate iritis and keratitis of the left eye. The

keratitis was considered to be tuberculous in origin. He had had recurring chronic attacks. The eye had almost never been entirely quiet. All standard forms of local treatment had been used, including a course of tuberculin therapy and rest in a sanatorium for five months. The patient had a seasonal vasomotor rhinitis and showed a sensitivity to ragweed and timothy pollen. In November 1934 a deep, yellow, discoid infiltrate developed in the cornea. This infiltrate progressed rapidly in size. On Oct. 22, 1935, there was moderate congestion. The corneal surface was smooth. The discoid infiltrate was fairly sharply

TABLE 6.—*Summary of Cases of Lipin Interstitial Keratitis (Group 5)*

No. of Case	Summary	Result
28	Rapidly increasing fat deposit in cornea for 16 months; arrested by vascular obliteration	Good
29	Tuberculous keratitis with fat deposit; present inflammation 1 year; arrested by vascular obliteration; slight recurrence of scleritis 1 month later; severe recurrence 16 months later	Good



Figs. 28-29.—Diagrams illustrating cases of lipin interstitial keratitis.

defined, practically filling the lower outer quadrant, measuring approximately 5 mm. in diameter (fig. 29 *A*). There was great vascularity, especially in the midstromal region. There were few cells in the aqueous and no deposits on the posterior corneal surface. The tension was normal. Vision was 18/200. The corneal vessels were obliterated with the diathermy needle, superficially from 2 to 7 o'clock and deeply from 4 to 6 o'clock (fig. 29 *B*). The stroma of the limbus was incised to approximately half its depth, obliterating most of the blood supply to the infiltrated area. On October 25 no subjective change was observed. The vessels showed no circulating blood. A fan of endothelial folds developed, extending radially outward from the infiltrated area (fig. 29 *C*). By November 8 definite subjective and objective improvement had taken place. On November 15 the patient was asymptomatic. The eye was white. During the week preceding December 6 the patient had a mild attack of scleritis in the left eye at 12 o'clock. On December 20 the eye was white, and the visual acuity was 20/100.

GROUP 6: TRACHOMATOUS KERATITIS

CASE 30.—A. La P., a man aged 21, was seen on Sept. 27, 1934. He gave a history of ocular irritation of four and a half months' duration. There was marked ptosis of the left upper lid. Examination showed slight congestion of the bulbar conjunctiva. The upper fornix was beefy red, with several large

translucent follicles and marked thickening of the conjunctiva. There was marked irregularity of the corneal surface, especially of the upper half, with a diffuse stippling, which stained lightly. In the upper and outer half of the cornea there was marked pannus formation. The lower limbus showed an early pannus (fig. 30 *A*). The right eye was normal. General physical examination gave negative results. The Wassermann reaction of the blood was negative. The intradermal reaction to 0.01 mg. of old tuberculin was positive. Roentgen examination of the teeth showed no apical abscesses. The pericorneal blood vessels were obliterated from 11 to 5:30 o'clock, local anesthesia being used. The diathermy needle was carried through the pannus at the limbus only (fig. 30 *B*). On October 5 the patient stated that there had been considerable improvement in symptoms. There was no corneal staining. Following the improvement immediately after operation a temporary recurrence of symptoms and a regrowth of vessels into the cornea to about the former amount took place. The patient was given applications of copper sulfate twice weekly from one week after operation for three months. On December 28 there was marked improvement in symptoms. The eye was practically white. The conjunctiva had cleared remarkably, and the patient was asymptomatic.

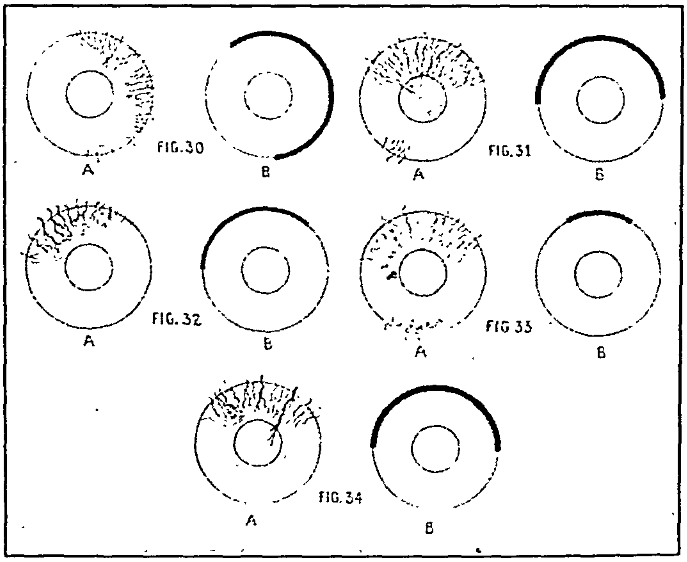
Case 31.—J. A., a professional wrestler aged 31, was seen on July 6, 1935. He contracted trachoma four years before and had had treatment of various kinds since then. Both eyes showed marked active trachoma with ptosis, conjunctival hypertrophy, follicles in both upper fornices and marked pannus above extending to the upper part of the pupillary area. The pannus was more marked in the left eye (fig. 31 *A*). During the two months preceding the present examination the patient had been treated regularly with a copper sulfate stick and drops of zinc sulfate. There had been some improvement of the right eye, but little of the left. Obliteration of the pericorneal blood vessels was carried out in the pannus of the upper half of the left eye (fig. 31 *B*). The diathermy needle was carried through the epithelium and Bowman's membrane of the limbus. The report of October 23 showed that there was marked and rapid improvement of the left eye after the operation. Treatment with copper sulfate was continued in both eyes. The right eye, which had not been peritomized, showed a marked lag in improvement. Peritomy was performed on the right eye on November 27. Owing to the patient's professional engagements in other cities, the postoperative course of the right eye could not be adequately followed.

Case 32.—K. La H., a woman aged 34, was seen on Nov. 6, 1934. She was born in Syria and undoubtedly contracted trachoma in childhood, although the diagnosis was not made until after she came to America in 1921. Treatment had been carried out by Dr. H. B. C. Riemer, who performed tarsectomy on each eye ten years previously. The patient was much improved by the operation but continued to have attacks of ocular inflammation. The Wassermann reaction of the blood was strongly positive. For a year the right eye had been constantly more inflamed than the left. There was slight congestion, and in the upper outer quadrant of the cornea there was a well marked pannus, at the border of which were several rough, faintly-staining infiltrates (fig. 32 *A*). The upper conjunctival fornix appeared fairly free from trachoma. Obliteration of the blood vessels was performed at the limbus from 9 to 2 o'clock, local anesthesia being used. The diathermy needle was carried through the blood vessels and epithelium (fig. 32 *B*). On Feb. 16, 1936, the patient said that she had been much improved by the operation. There was little pain after operation. Within two weeks the eye became white and quiet. There was some return of blood vessels in the upper and outer quadrants of the cornea, but the scars decreased in density, as did the roughness of the epithelium.

CASE 33.—L. McM., a girl aged 6 years, was seen on Aug. 3, 1934. She had had constant inflammation of both eyes for the preceding two and one half years. A definite diagnosis had never been made. Both corneas showed superficial punc-

TABLE 7.—*Summary of Cases of Trachomatous Keratitis (Group 6)*

No. of Case	Summary	Result
30	Active trachoma; marked but incomplete improvement by vascular obliteration; also received copper sulfate	Fair
31	Active trachoma; marked improvement after vascular obliteration; also received copper sulfate	Good
32	Chronic trachoma with periodic exacerbations—keratitis; considerable improvement after vascular obliteration	Good
33	Active trachoma in child, 2½ years' duration; marked improvement after vascular obliteration	Good
34	Chronic trachoma with moderate pannus; no definite improvement after vascular obliteration	No improvement



Figs. 30–34.—Diagrams illustrating cases of trichomatous keratitis.

tate staining and small areas of infiltration, particularly in the upper half of each cornea. A well marked but thin pannus entered the pupillary space from above in each eye. The conjunctivas were moderately congested, and both upper lids were beefy red with many translucent follicles. The patient had been admitted to the ward for contagious diseases several times, where various forms of treatment had been administered with limited success. The inflammation responded best to the regular application of crystals of copper sulfate. For a month before the present examination there had been a marked exacerbation of the symptoms, especially in the right eye. The pannus and areas of densest infiltration in the right eye are shown in figure 33 *A*. Obliteration of the pericorneal blood vessels from 10 to 2 o'clock was performed. The diathermy needle was carried well through the epithelium of the limbus (fig. 33 *B*). A biopsy specimen was taken from the upper fornix for pathologic study. This showed "follicular hyperplasia consistent with trachoma." Examination on August 17 showed that there had been marked improvement in the appearance of the eye during the preceding week. The cornea did not stain. The epithelium was smoother, and the vessels much less conspicuous. A report on May 16, 1935, stated that the right eye had

been distinctly the less troublesome one during the past year. It exhibited less pannus, and the corneal appearance was better in every way. The patient continued treatments with copper sulfate in the outpatient department.

CASE 34.—S. Di. M., a man aged 25, was seen on Nov. 9, 1935. He was born in Italy and had had symptoms of trachoma since adolescence. No treatment was given until six weeks before the present examination, when he was admitted to the Massachusetts Eye and Ear Infirmary. Examination showed trachoma of both eyes, more active in the left. There were scarring of the tarsi and thickening of the conjunctiva of both upper fornices with a few scattered lymphoid follicles. Both eyes showed pannus and corneal scars, the left more marked (fig. 34 *A*). Applications of copper sulfate were given, with a slow response, particularly in the left eye. Obliteration of the pericorneal blood vessels was performed in the upper half of the left cornea. The diathermy needle was carried through the epithelium only (fig. 34 *B*). An uneventful convalescence followed the operation. By June 1, 1936, the congestion had diminished until it was approximately as it was before operation. There may have been some improvement in the appearance of the cornea, although it was not marked. There was a return of vascularization to approximately its previous amount. The visual acuity remained unchanged.

GROUP 7: HYPOPYON ULCER

CASE 35.—W. F., a man aged 33 (a patient of Dr. Paul Haire), during March 1935 was struck in the left eye by a piece of ice. The eye was sore for one week and continued to be irritated and slightly congested. A report on July 3 stated that the eye had been sore for the preceding four weeks. There was marked congestion. An oval ulcer, 3 by 4 mm., was present in the upper inner quadrant of the cornea. There was superficial vascularization (fig. 35 *A*). A hypopyon ulcer measuring 1 mm. was present. A direct smear was negative for organisms. Inoculation of a rabbit for herpes gave negative results. On July 9 the patient was admitted to the Massachusetts Eye and Ear Infirmary. No improvement occurred on conservative treatment. The ulcer advanced into the pupillary space (fig. 35 *B*), and the vascularization increased. Although the hypopyon had disappeared, the aqueous was full of cells. The visual acuity was 2/200. A smear showed rare pneumococcus. Inoculation of a rabbit for herpes was again negative. The corneal vessels were obliterated from 9 to 12 o'clock with a diathermy needle (fig. 35 *C*). On July 12 it was found that there had been no advance in the ulcer since operation. The patient was discharged from the infirmary. On July 22 there was definite improvement, only slight congestion remaining. The ulcer was much smaller. On July 25 there was no staining of the ulcer. On August 3 the eye was completely white. On August 29 the patient was asymptomatic.

GROUP 8: UNCLASSIFIED ULCER

CASE 36.—P. F. McN., a man aged 59, was first seen on Oct. 25, 1937, complaining of pain in the left eye for three days. There were moderate irritation and marked congestion. An oval ulcer was found in the cornea near the limbus at 1 o'clock, measuring approximately 2 by 3 mm. in size (fig. 36 *A*). There were slight infiltration of the surrounding cornea and moderate secondary iritis. A subconjunctival injection of epinephrine was given to complete pupillary dilatation, and atropine, fomentations and a bandage were prescribed. A careful search for focal infections showed marked peridental disease. The Wassermann reaction of the blood and also the reactions to tuberculin were negative. The patient

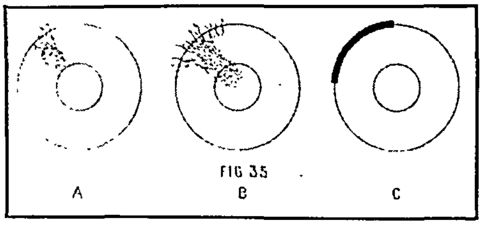


Fig. 35.—Diagram illustrating a case of hypopyon ulcer.

TABLE 8.—*Summary of Case of Hypopyon Ulcer (Group 7)*

No. of Case	Summary	Result
35	Ulcer following injury, 4 months; rare pneumococcus in direct smear; small hypopyon; rapid and complete improvement after vascular obliteration and hospital care	Excellent

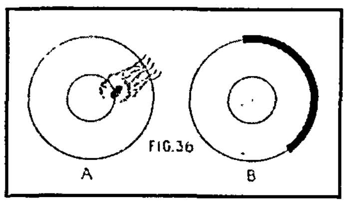


Fig. 36.—Diagram illustrating a case of unclassified ulcer.

TABLE 9.—*Summary of a Case of Unclassified Ulcer (Group 8)*

No. of Case	Summary	Result
36	Advancing ulcer, 15 weeks' duration; promptly arrested after vascular obliteration	Excellent

TABLE 10.—*Summary of Results in All Groups*

Groups	Excellent	Good	Fair	Unimproved
Group 1: dendritic keratitis (keratitis metaherpetica).........	4	2	0	0
Group 2: ocular rosacea	3	0	3	1
Group 3: primary superficial keratitis, probably tuberculous..	2	2	5	3
Group 4: keratitis following trauma...........................	1	0	0	1
Group 5: lipin interstitial keratitis	0	2	0	0
Group 6: trachomatous keratitis	0	3	1	1
Group 7: hypopyon ulcer	1	0	0	0
Group 8: ulcer unclassified	1	0	0	0
Totals..	12	9	9	6

made regular visits to the clinic, and most of the usual remedies were tried. The ulcer showed no tendency to heal. It remained about the same size, but its location on the cornea gradually became more central, until on Feb. 2, 1938, fifteen weeks after onset, it was located about 4 mm. from the limbus. The ulcer was followed by many superficial blood vessels and a few in the midstroma of the cornea. Photographs were taken, and on February 2 the superficial blood vessels were obliterated at the limbus from 12 to 5 o'clock (fig. 36 *B*). The eye showed striking improvement in two days, with less staining of the ulcer. Several folds became apparent in the posterior corneal surface and persisted until the seventh day. The ulcer then showed no staining, and two weeks after the operation the eye was completely white and quiet.

SUMMARY AND CONCLUSIONS

The effect of interrupting the blood vessels at the limbus has been observed in 36 cases of various types of keratitis associated with vascularization of the cornea. Considering the chronic nature of the disease in each case, the results were good and leave no doubt that peritomy is of great value in certain corneal conditions.

The assumption that the newly formed blood vessels cause and maintain the reactivation of the lesions explains the results obtained in these cases.

Corneal blood vessels may not only cause opacification but necrosis and an actual increase in the amount of corneal infiltration.

The metaherpetic ulcer is probably due to a nutritional disturbance or to some noxious substance brought to the cornea by the abnormal blood vessels. Obliteration of the pericorneal blood vessels is particularly valuable for this condition.

The so-called wandering phlyctenule of the cornea is not always tuberculous in origin. A similar condition may result from herpes corneae and other causes.

Complete peritomy does not cause any visible disturbance in corneal epithelium. Almost all of the pericorneal blood supply can be excluded without harmful effect. In view of this and other experimental evidence cited and in view of the fact that tears are rich in sugar and nitrogenous substances, it is probable that the epithelium derives its nutrients chiefly from the lacrimal secretion and utilizes oxygen directly from the surrounding air.

ABSTRACT OF DISCUSSION

Dr. Albert D. Ruedemann, Cleveland: Vascularization has come to be thought of as a healing process, and it is sometimes difficult to believe that blood vessels themselves may be the cause of most of the pathologic changes so far as the scarring is concerned. I am not so certain but that it might not be advisable in certain instances to stop the progress, even during the active stage. I do not feel that vascularization is always necessary in order to secure good healing. Whenever vascularization begins, scar tissue forms, and scar tissue cannot be

removed. If it were possible to stop the ingrowth of scar tissue from the limbus and to bring the healing process in from the corneal side, then there would be a better chance of obtaining a good clear cornea. It has been my experience in studying the conjunctival capillaries that frequently the deep limbal capillaries will grow up from below and back to the limbus rather than from the limbus to the lesion. In these cases it is not possible to stop the progress of the blood vessels as has been done in cases of lime burn with pseudopterygia. Dr. Gundersen has reported good results in cases in which he has cauterized the vessels rather deeply.

For some time I have felt that if it were possible to decrease the congestion of the ocular conjunctiva in order to limit the amount of blood flowing through the superficial structure of the cornea, the formation of some of these corneal scars might be prevented.

I feel as does Dr. Gundersen, that the healing process followed by vascularization and the formation of scar tissue might be better regulated by cutting out some of the larger vessels. I believe that more scars form during the reparative processes because of a blood vessel, and that if some method is found which will control the formation of blood vessels the amount of scarring might be controlled.

I should like to add one more example of vascularization as a factor in hindering a good result, as exemplified by the filtering operation for glaucoma at the limbus. It has been noted that those patients who have marked vascularization or who have congestion at the time of operation are likely to have a less satisfactory surgical result than those patients who have a noninflammatory, noncongestive or plain chronic simple glaucoma.

I have been studying these problems in the hope of finding out whether the corneal scarring is due to an inhibition to the formation of blood vessels by the aqueous as it filters through the trephine opening or the inclusion opening, whether it is due to stimulation of the formation of blood vessels by the irritation set up at the time of the operation or whether it is due to an irritative aqueous, such as is found in some of the cases of chronic inflammation.

I am certain that those patients who have the best results are those who have the fewest blood vessels at the time of operation, without reaction and without formation of new blood vessels.

Dr. Charles A. Bahn, New Orleans: There are approximately 10,000 people in the United States who are blind from injuries or disease of the cornea. Dr. Gundersen has not presented an improvement in the treatment of some of the conditions which are responsible.

The formation of new blood vessels in the cornea represents a chemical response of a certain character of which but little is known. This formation varies in persons, in diseases and under other conditions. This diversity in the reaction is illustrated in the wide variations of pannus that exist in cases of trachoma.

Owing to the specialization of the cornea, the same blood vessel formation which is essential to healing in most parts of the body sometimes becomes a menace in the cornea. Here, there are two types of newly formed blood vessels, superficial and deep. The superficial grow from the limbal loops, and their purpose is apparently to facilitate

regeneration of the epithelial cells. These new blood vessels, however, may contain noxious substances which not only poison the epithelial cells but also poison more or less permanently focal areas in Bowman's membrane on which the baby epithelial cells grow. These baby epithelial cells are born sick, and they die sick as they advance to the exterior. The areas in which they live are sick areas, and thus there are advancing zones in some corneal conditions, such as marginal ulcerative keratitis, rodent keratitis, wandering phlyctenules, etc. It is possible that this same principle is involved in the formation of pterygia. Vascular obliteration in this group of conditions apparently finds its greatest field of usefulness.

In the deep blood vessels which I believe grow principally from the scleral vessels, the process is apparently different. These blood vessels bring nutritive and protective substances as well as cellular elements to the mesodermic cornea. It is the fibroblasts which they bring that become a menace to healing.

The production of fibroblasts varies with different diseases and in different persons. For example, in cases of vernal conjunctivitis the disease may be present for years with practically no cicatrization, while in some cases of trachoma and of pemphigus cicatrization soon becomes the dominant factor in the tragic picture.

In deep vascularization obliteration is often desirable to reduce cicatrization and possibly delayed healing. In acute infectious ulcers, such as pneumococcic ulcers, vascular obliteration is generally of little value.

Dr. Gundersen's method of using vascular obliteration is superior. The delimiting keratotomy of Gifford largely depends on vascular obliteration. Peritomy is also a more or less crude application of the same principle.

Dr. Gundersen's interesting contribution marks a step forward in the treatment of some corneal diseases that often lead to blindness.

Dr. Trygve Gundersen, Boston: It must occur to all after hearing this paper that I do not favor the use of vasodilators, particularly ethylmorphine hydrochloride. I have found them ineffective in treating corneal lesions. On the other hand, the use of vasoconstrictors has often been of great value, with the exception of those vasoconstrictors which are followed by a compensatory vasodilatation, such as epinephrine and ephedrine. The value of zinc sulfate in the treatment of many corneal and conjunctival diseases, aside from its action as an antiseptic, may well lie in its effect as an astringent or a vasoconstrictor. An ointment which has been found to be effective at the Massachusetts Eye and Ear Infirmary is an old German preparation consisting of: zinc oxide, 7.8 Gm., or 2 drachms; ichthammol, 0.233 Gm., or 3½ grains; white petrolatum, 23.4 Gm., or 6 drachms. It is important that the ingredients be reduced to a fine impalpable mixture.

CONGENITAL GROUPED PIGMENTATION OF THE RETINA

REPORT OF A CASE

CHARLES A. PERERA, M.D.
NEW YORK

Congenital grouped pigmentation of the retina presents an uncommon but characteristic ophthalmoscopic picture, which was well described by Stephenson [1] in 1891. This author, in reporting 3 cases, said: "The changes, which occupy a sector-like portion of the fundus, consist of groups made up of black to dark chocolate-brown spots, which, as seen by the direct method, range in size from ½ to 2 mm. or more; in general terms it may be said that the farther the spots lie from the optic disc the larger they become."

A study of the cases reported in the literature shows that the clinical picture of this entity, which has also been called melanosis of the retina and nevoid pigmentation of the fundus, includes certain characteristic findings: 1. The condition is unilateral and more frequent in males than in females. 2. There is no hereditary aspect, and consanguinity does not play a role. 3. There is no association with congenital deformities or with other changes (inflammatory or degenerative) in the fundus or other parts of the eye. 4. The condition is stationary. 5. The fundus is involved in sectors, leaving the macula usually free of lesions. 6. The pigment spots lie deep to the retinal vessels. 7. The visual fields and acuity are not affected. 8. Night blindness is not present in the affected eye.

An early drawing of a typical case is to be found in the atlas of Jaeger,[2] published in 1869. The literature has been thoroughly reviewed by Höeg,[3] Blake,[4] Schieck [5] and Mann.[6] Other authors who have

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1. Stephenson, S.: A Peculiar Form of Retinal Pigmentation, Tr. Ophth. Soc. U. Kingdom **11**:77-82, 1891.

2. Jaeger, E.: Ophthalmoskopischer Hand-Atlas, Vienna, 1869, pp. 126-127 and plate XVI, fig. 76.

3. Höeg, N.: Die gruppierte Pigmentation des Augengrundes, Klin. Monatsbl. f. Augenh. **49**:49-77, 1911.

4. Blake, E. M.: Congenital Grouped Pigmentation of the Retina, Tr. Am. Ophth. Soc. **24**:223-233, 1926.

5. Schieck, F.: Die Erkrankungen der Netzhaut, in von Schieck, F., and Brückner, A.: Kurzes Handbuch der Ophthalmologie, Berlin, Julius Springer, 1930, vol. 5, pp. 588-590.

6. Mann, W. A., Jr.: Grouped Pigmentation of the Retina, Arch. Ophth. **8**:66-71 (July) 1932.

reported cases or have discussed the condition include Jacobi,[7] Dodd,[8] Parsons,[9] Chance,[10] Leber,[11] Kraupa,[12] Hedde,[13] Holm,[14] Collins and Mayou,[15] Bedell [16] and Ida Mann.[17]

I am unable to find a report of the microscopic study of an involved eye. The lesion may be due, as Ida Mann has suggested, to localized patches of pigment developing abnormally in the inner layer of the optic cup or to localized proliferations of the pigment epithelium forming the outer layer of the optic cup.

The condition must be distinguished from congenital solitary pigment masses in the fundus and from the patches of pigment following inflammatory and vascular disease of the eye.

Appearance of right fundus, showing congenital grouped pigmentation of the retina.

Although many cases have been described, the extent of involvement of the fundus in the case reported here makes it worthy of record in the literature.

REPORT OF CASE

M. S., a 4 year old asthmatic Jewish boy, was brought to the Vanderbilt Clinic in November 1933, because of symptoms due to mild conjunctivitis. Examination under cycloplegia with atropine showed vision of 20/30— and hypermetropia of 2 diopters. The right fundus contained many black and dark gray pigmented spots of varying sizes lying beneath the retinal vessels; these spots had well defined rounded or angular margins. The left fundus was normal. The parents had normal fundi.

7. Jacobi, J.: Pigmentmassen in der Retina ohne Störung der Sehfunctionen, Arch. f. Ophth. **14**:144-147, 1868.

8. Dodd, H.: Congenital Pigmentation of the Retina, Tr. Ophth. Soc. U. Kingdom **15**:194, 1895.

(Footnotes continued on next page)

This case was presented before the New York Ophthalmological Society in 1934 (by invitation), as an example of congenital grouped pigmentation of the retina.

The boy was seen again in April 1938, with complaints referable to conjunctivitis. Under cycloplegia with homatropine hydrobromide, vision in each eye was 20/20, the patient accepting in the right eye a + 1.00 sph. combined with a + 0.37 cyl., ax. 170, and in the left eye a + 0.75 sph. The fundi were unchanged as compared with the findings of 1933. The appearance of the pigment spots in the right eye is well depicted in the accompanying photograph of a drawing by M. Quinlan. The visual fields were normal; no scotomas could be plotted to correspond with any of the pigmented lesions.

70 East Sixty-Sixth Street.

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9. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1906, vol. 3, p. 904.

10. Chance, B.: Concerning Two Examples of "A Peculiar Form of Retinal Pigmentation" (Stephenson), Ophth. Rec. **24**:186-188, 1915.

11. Leber, T., in von Graefe, A., and Saemisch, E. T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, pp. 2041-2044.

12. Kraupa, E.: Studien über die Melanosis des Augapfels, Arch. f. Augenh. **82**:67-93, 1917; Beiträge zur Morphologie des Augenhintergrundes III, Klin. Monatsbl. f. Augenh. **67**:20-21, 1921.

13. Hedde, C.: Gruppenförmige naevoide Pigmentierung der Netzhaut, Klin. Monatsbl. f. Augenh. **64**:301-306, 1920.

14. Holm, E.: Zwei Fälle von gruppierter Pigmentierung, Klin. Monatsbl. f. Augenh. **67**:451-454, 1921.

15. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925, p. 27.

16. Bedell, A. J.: Some Anomalies of the Fundus, Tr. Sect. Ophth., A. M. A., 1931, pp. 303-337.

17. Mann, I.: Developmental Abnormalities of the Eye, New York, The Macmillan Company, 1937, pp. 153-154.

RETINAL ARTERIOLAR CHANGES AS PART OF AN INDUCED GENERAL VASOSPASTIC REACTION

EFFECT OF TOBACCO AND COLD

PAUL L. CUSICK, M.D.

AND

WALLACE E. HERRELL, M.D.

ROCHESTER, MINN.

Functional vasoconstriction, affecting mainly the arteriolar portion of the vascular system, is now generally conceded to play a major part in the production of the peripheral resistance in cases of essential hypertension. The lack of organic change in the arterioles of some hypertensive patients and the drop in blood pressure often seen after the administration of vasodilating and sedative drugs and after section of the splanchnic and other sympathetic nerves, as well as the marked fluctuations in blood pressure in the same persons at various times, all suggest this to be true.

The generalized narrowing observed ophthalmoscopically of the retinal arterioles of patients with the various forms of diffuse vascular disease associated with hypertension is regarded by many as due to spastic constriction or increased tonus in the arterioles rather than to organic change. The recent measurements of the caliber of the retinal arterioles in health and in disease made by Lobeck [1] show graphically the reduction in caliber which is present in cases of vascular disease and hypertension. With his instrument the average caliber of the retinal arterioles of healthy persons varied between 134 and 88 microns. There was a gradual reduction in caliber in various types of hypertensive disease until in patients with nephrosclerosis associated with retinitis, the caliber varied between 33 and 26 microns.

It occurred to us that ophthalmoscopic examination of the retinal arterioles and measurement of their caliber before and during an induced generalized vasoconstrictive reaction associated with a rise in systemic

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From the Section on Ophthalmology (Dr. Cusick) and the Division of Medicine (Dr. Herrell), the Mayo Clinic.

Abridgment of thesis submitted by Dr. Cusick to the Faculty of the Graduate School of the University of Minnesota in partial fulfilment of the requirements for the degree of M.S. in Ophthalmology.

1. Lobeck, E.: Ueber den Durchmesser der Netzhautgefässe am gesunden und kranken Menschen, Arch. f. Ophth. **136**:434-456 (Feb. 20) 1937.

blood pressure might yield information on three points: (1) whether the arterioles of the retina are supplied with vasomotor nerves, (2) whether these arterioles take part in a vasomotor response to a peripheral or toxic stimulus and (3) whether this response, if it occurs, is in the nature of a generalized narrowing or a localized spastic constriction.

One of us was interested at the time in a group of patients who had an idiosyncrasy to tobacco. After these persons smoked one or two cigarets a reaction occurred which was characterized by a marked rise in the systolic and diastolic blood pressure, an increase in the pulse rate and a drop in the cutaneous temperature of the fingers and toes, indicative of peripheral vasoconstriction. This reaction was much more marked with some brands of cigarets than with others and was not affected by any of the filters commonly used. These patients were all hyperreactors to the cold pressor test of Hines and Brown,[2] and the rise in blood pressure after smoking closely paralleled the rise during the cold pressor test (fig. 1). This condition is by no means rare, as one of us (Herrell) has collected reports of a number of cases during the last year and 2 reports have previously been made dealing with the cases from a medical standpoint.[3]

Five of the patients in this group were subjected to ophthalmoscopic examinations, and measurements of the retinal arterioles were made both before and after smoking, a modified Morgan graticule in a Keeler ophthalmoscope being used. With this method a number of squares are reflected on the retina, each square measuring 100 microns on a side. Measurements were made of the four main arteriolar branches in each eye about ½ disk diameter from the optic nerve head, and the average measurement was then calculated. By this method the average caliber of normal retinal arterioles varied between 50 and 60 microns for the nasal arterioles and between 100 and 120 microns for the temporal arterioles.

On repeated examination of all 5 patients a definite measurable reduction in the caliber of the retinal arterioles was found to take place after smoking. Typical average measurements are given in table 1. The narrowing was generalized rather than localized in any particular arteriole, although it varied in different arteriolar branches and in the two eyes of the same subject. This generalized narrowing of the arterioles was similar to that seen in the early phases of increasing blood

2. Hines, E. A., Jr., and Brown, G. E.: The Cold Pressor Test for Measuring the Reactibility of the Blood Pressure: Data Concerning Five Hundred and Seventy-One Normal and Hypertensive Subjects, Am. Heart J. **11**:1-9 (Jan.) 1936.

3. Herrell, W. E.: Idiosyncrasy to Tobacco: Report of a Case, Proc. Staff Meet., Mayo Clin. **13**:1-6 (Jan. 5) 1938. Herrell, W. E., and Cusick, P. L.: Vascular and Retinal Abnormalities Following Inhalation of Tobacco Smoke: Preliminary Report, ibid. **13**:273-279 (May 4) 1938.

pressure in the hypertensive toxemia of pregnancy. Localized spastic narrowing, such as is seen in the later phases of the toxemia of pregnancy, was not observed in any case. Photographs of the fundus were made in several cases before and after smoking (fig. 2).

The retinal diastolic pressure of all patients was measured by Baillart's dynamometer before and after smoking. As was to be expected,

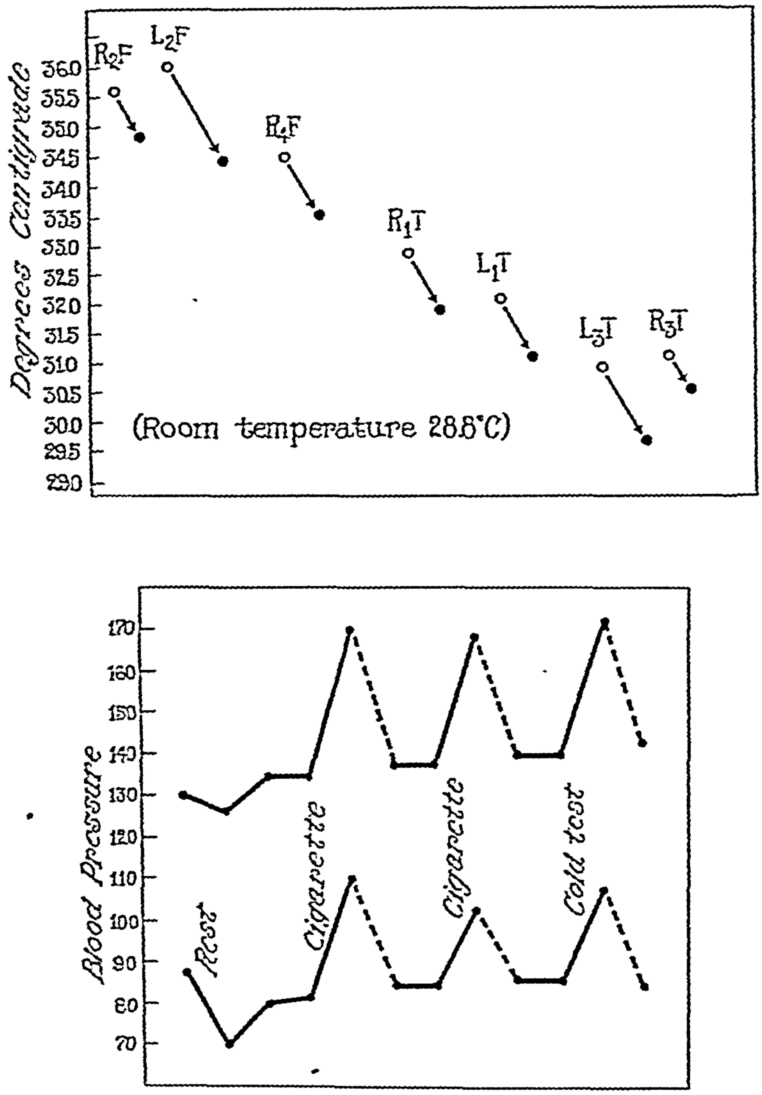

Fig. 1.—The charts showing the cutaneous temperature of the various digits and the blood pressure of a patient with an idiosyncrasy to tobacco before and after smoking a cigaret; also his reaction to the cold pressor test. In the upper chart the cutaneous temperature before smoking a cigaret is indicated by the circle and that after smoking by the black dot.

an elevation of this pressure occurred in all patients proportionate to the elevation in systemic blood pressure. When the retinal diastolic pressure was higher in one eye than in the other, there was, as a rule, a more measurable reduction in caliber in the arterioles of the eye with the higher diastolic pressure. This was particularly true of 1 patient.

Measurements of intraocular tension were made with the Schiötz tonometer for all patients before and after smoking. A slight but definite increase in tension occurred in all 5, the rise varying between 1 and 3 mm. of mercury and averaging 2 mm. This increase appeared to be associated with the increase in intraocular arterial blood pressure. It did not compare directly with the diminution in caliber of the arterioles. The eye with the greater diminution in arteriolar caliber showed at times a lower intraocular tension than the opposite eye.

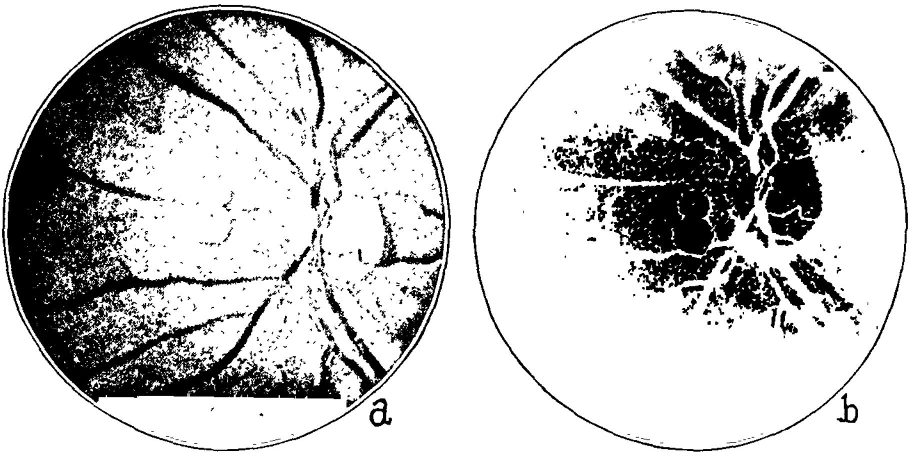

Fig. 2.—The ocular fundus of a patient with an idiosyncrasy to tobacco; *a*, before and *b*, after the smoking of a cigaret.

TABLE 1.—*Average Arteriolar Caliber Before and After Smoking in Patients with an Idiosyncrasy to Tobacco*

Patient	Age, Years	Average Caliber of 8 Arterioles, Microns		Reduction in Caliber, per Cent
		Before Smoking	After Smoking	
1	57	88	72	18.2
2	23	92	68	26.1
3	66	110	88	20.0
4	35	120	90	25.0
5	43	70	56	20.0

Since the vasomotor reaction of the patients who showed an idiosyncrasy to tobacco was similar to that observed with the cold pressor test, an attempt was made to study the changes in the retinal arterioles of subjects undergoing the cold pressor test. In this test, as described by Hines and Brown, a basal level of blood pressure is established by a period of rest and one hand is then immersed up to the wrist in ice water at 4 C. The blood pressure is taken at the end of thirty seconds and again at the end of sixty seconds. The majority of persons show some elevation of both the diastolic and the systolic pressure. Patients

with latent hypertension show a more definite elevation, and patients with established hypertension show a still more marked rise in blood pressure, depending considerably in degree on the severity of the disease. Hines and Brown expressed the belief that this increase in blood pressure was produced by a neurogenic reflex, as the application of a tourniquet above the wrist did not affect the reaction and as they had found that the reaction was also produced in previously adrenalectomized dogs.

Measurements were made of the caliber of the retinal arterioles of 25 patients undergoing the cold pressor test. It was found to be

TABLE 2.—*Changes in the Caliber of the Retinal Arterioles of Persons During the Cold Pressor Test*

Patient	Blood Pressure, Mm. of Hg		Average Caliber of Arterioles, Microns	
	Before Test	During Test	Before Test	During Test
1	130/ 90	150/110	90	68
2	112/ 68	138/ 88	80	72
3	110/ 60	126/ 78	72	60
4	104/ 76	124/ 96	80	66
5	90/ 60	124/ 90	96	76
6	112/ 70	130/ 98	82	72
7	130/ 85	170/110	104	84
8	110/ 68	128/ 96	120	120
9	110/ 70	138/ 94	100	100
10	118/ 70	126/ 98	76	60
11	120/ 80	135/100	84	70
12	92/ 68	118/ 88	70	56
13	150/120	165/140	60	52
14	114/ 72	142/108	74	52
15	110/ 80	120/ 98	82	66
16	110/ 62	130/ 84	90	72
17	110/ 80	115/ 90	104	104
18	116/ 70	168/110	72	52
19	132/ 90	146/105	90	74
20	100/ 64	122/ 88	80	70
21	115/ 82	124/ 84	96	96
22	130/ 95	154/130	58	52
23	120/ 90	130/105	68	68
24	130/ 88	150/112	90	72
25	140/ 90	180/105	90	74

more difficult to make satisfactory measurements for this group of persons than for the former group because of the time element. The blood pressure usually returns rapidly to normal after the hand is removed from the ice water, and, because of the pain occasioned, it is difficult for the patient to keep his hand in the water for more than from one and a half to two minutes. For this reason only two arterioles (the superior and inferior temporal) were measured. Twenty of the 25 patients, however, showed a measurable reduction in arteriolar caliber during the test, the same uniform narrowing being observed as for the patients who were sensitive to tobacco. Three of the 5 remaining patients showed only slight or negligible rise in systemic blood pressure during the test. It is probable that if more arterioles of the other 2 patients had been measured a change would have been noted in some of them, in spite of a rise in blood pressure. The blood pres-

sure readings and arteriolar measurements are shown in table 2. Neither the retinal arterial blood pressure nor the ocular tension was studied for this group of patients.

Of this second group of patients, 19 had basal blood pressures which were considered normal; the retinal arteriolar measurements varied from 70 to 120 microns and averaged 86 microns, which is definitely lower than the normal average. Sixteen of the 19 patients were hyperreactors to the cold pressor test. The finding of retinal arterioles of smaller caliber in this group is interesting because it may furnish additional evidence that these patients had latent hypertension. The caliber of the retinal arterioles of the 2 patients who definitely were not hyperreactors was well within the normal average; the arterioles of 1 patient measured 96 and of the other 104 microns. The 1 remaining patient who showed an average retinal arteriolar caliber of 82 microns was a questionable hyperreactor; his diastolic and systolic pressures both rising 18 mm. of mercury.

The measurements of the retinal arteriolar caliber of the 6 patients with definite hypertension varied between 58 and 90 microns and averaged 76 microns. This agrees with Lobeck's findings that when hypertension is established there is a definite reduction in the retinal arteriolar caliber below the average normal.

SUMMARY

Five patients with an idiosyncrasy to tobacco showed a reduction in caliber of the retinal arterioles after smoking which varied between 18.2 and 26.1 per cent and averaged 20.8 per cent. In 20 of the patients who showed changes in retinal arteriolar caliber during the cold test, the reduction in caliber varied between 10 and 28.6 per cent and averaged 18.6 per cent.

An average rise in intraocular tension of 2 mm. of mercury was noted for the patients who showed a vasospastic reaction from smoking. This rise was variable, however, and it is evident that there was no causative relation between the slight rise in tension and the narrowing of the arterioles.

A rise in the retinal arterial diastolic pressure occurred, as would be expected, with a rise in the systemic blood pressure. One patient with a rather marked variation between the diastolic pressure in the two eyes after smoking showed a more measurable diminution in caliber in the eye with the higher diastolic pressure.

It seems justifiable to conclude from these studies that an elevation in blood pressure induced by the methods employed is associated with a narrowing of the caliber of the peripheral arterioles which also involves the arterioles of the retina. As far as can be judged from ophthalmoscopic appearances, the narrowing of caliber is uniform

throughout the course of any individual arteriole but is not present in equal amounts in all the arterioles. The character of the narrowing suggests that it is due to increased vasomotor tonus rather than to active angiospasm. If our observations on these patients are correct, it seems necessary to assume that the arterioles of the retina are supplied with vasomotor nerves and can undergo active vasoconstriction.

COMMENT

The almost constant association of a transitory reduction in caliber of the retinal arterioles with the rise in systemic blood pressure accompanying the general vasopressor action of cold and tobacco suggests that the generalized narrowing of the retinal arterioles observed ophthalmoscopically in many cases of hypertensive disease (and definitely demonstrated by the measurements of Lobeck) is due primarily to active vasoconstriction or increased vasomotor tonus rather than to actual structural change in the walls of the vessels. This constriction probably occurs through the mechanism of the sympathetic nervous system. If this is true, it would be possible, theoretically, for the caliber of previously narrowed retinal arterioles to increase in hypertensive disease under conditions which would lessen vasopressor stimuli. Such relaxation has been observed clinically in a number of cases in which the systemic blood pressure has fallen after operations on the sympathetic nervous system or after other methods of treatment. The relaxation occurs at times spontaneously in cardiac failure and in other conditions which reduce vasomotor tonus.

HERPES ZOSTER OPHTHALMICUS

REPORT OF A CASE

FERDINAND L. P. KOCH, M.D.
Fellow in Ophthalmology, the Mayo Foundation
ROCHESTER, MINN.

Justification for reporting the following typical case of epidemic herpes zoster ophthalmicus lies in the fact that the patient was 5½ years of age. The earliest age at which any patient with this disease had been seen theretofore at the Mayo Clinic was 12 years. The attack occurred on the left side in that instance, and there was no involvement of the nasociliary nerve, nor were there any of the more infrequent complications. It is well known that the disease almost exclusively affects adults, including the aged; however, several cases have been reported in which the patients were children,[1] but it is remarkable how few such reports can be found on search of the literature. This is especially noteworthy when one considers the reported clinical evidence associating herpes zoster with chickenpox.[2]

REPORT OF CASE

A white boy 5 years and 6 months of age, of Irish-American descent, was seen at the clinic and immediately was hospitalized on April 22, 1938, four days after the onset of mild fever, accompanied by several light chills. There was also severe neuralgic pain over the area supplied by the ophthalmic branch of the left trigeminal nerve. This was coincident with the appearance of typically distributed, small, clear, fluid-filled vesicles. There was no history of preceding infection except that a small hordeolum of the left lower lid had appeared twelve days prior to the onset of the herpes. Spontaneous evacuation had occurred, with complete subsidence, four days later. The mother had given the child 20 grains (1.3 Gm.) of acetylsalicylic acid daily for three days before calling a physician; in addition, very hot, moist compresses had been applied almost continuously for the same period over the affected areas.

Dermatologic examination revealed typically demarcated, erythematous herpetic dermatitis with many crusted lesions over the distribution of the ophthalmic branch of the left trigeminal nerve, including the nasociliary branch on that side. The lesions extended only to the midline. There were many large bullae over the frontal region, laterally; they were filled with a dark yellowish, turbid fluid and ruptured in a few hours.

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1. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, pp. 518-519.

2. Amies, C. R.: The Elementary Bodies of Zoster and Their Serological Relationship to Those of Varicella, Brit. J. Exper. Path. **15**:314-320 (Oct.) 1934.

Ophthalmologic examination was difficult to accomplish because of the marked hyperalgesia, hyperemia and edema of the left upper lid, which overrode the lower lid by 1 cm.; however, retraction revealed the presence, just within the margin of the lid, of three small, true conjunctival herpetic lesions of the palpebral surface of the upper lid in its medial third. There was marked redness and injection of the palpebral and bulbar conjunctiva, and the latter was the site of marked chemosis. The cornea was clear. The anterior chamber was normal in depth, and the pupil was constricted, but round and regular. Other observations could not be made at this time. The right eye was essentially normal throughout.

Daily examination by members of the section on pediatrics revealed nothing remarkable until the fifth day after the patient's admission to the hospital, at which time the generalized vesicular eruptions of a mild attack of varicella manifested themselves. The dermatitis of herpes had improved markedly, and no more bullae had appeared. Ophthalmologically, the right eye was essentially normal throughout except for some mild edema of both lids. Vision was 6/5 without correction; the visual field was roughly normal, and the intraocular tension, determined by means of the Gradle-Schiötz tonometer, was 9 mm. of mercury.

Vision of the left eye was 6/10 without correction; the visual field was roughly normal, and the intraocular tension was 6 mm. of mercury. The margins of the lids once more approximated each other, although considerable mild induration was still present and interfered with full physiologic widening of the palpebral fissure. The palpebral and bulbar conjunctival surfaces remained moderately edematous and injected, and the herpetic bulbar conjunctival ulcers presented necrotic bases. The external ocular movements were normally performed. There was no evidence of intraocular inflammation. Corneal sensitivity was lost. The cornea was clear except for seven small, round areas nasally, of superficial grayish infiltration of the substantia propria. These were typical of herpes zoster keratitis and did not stain with fluorescein. The largest was just less than 3 mm. in diameter. The pupil was now dilated and fixed at 5 mm. Its margin was slightly irregular, and it did not react to any of the usual stimuli; however, it dilated readily and well with 1 per cent solution of homatropine hydrobromide. The anterior chamber was of normal depth, and the media were clear. The fundus was essentially normal to ophthalmoscopic examination except for minimal, generalized, arteriolar narrowing secondary to the mild generalized ischemic pallor of the nerve head. The subjacent retina was slightly edematous, and many fine and small, coalescent, yellowish white exudates were scattered centrally about the macular area except on its temporal side. The exudates were thought to have appeared subsequent to the massive edema of the lids and the orbital tissues.

On the patient's admission to the hospital, an ointment containing 3 per cent ichthammol was applied to the affected areas of the skin, and this was followed, after the first day, by continuous application of calamine lotion packs. Mild protein silver solution, 25 per cent, was instilled three times daily into the left eye. General treatment was begun with the intramuscular injection on the day of admission of 6 cc. of whole blood. This amount was administered daily for five days. The patient remained comfortable under the oral administration of pentobarbital sodium, ¾ grain (0.05 Gm.), twice a day. The temperature began to approach normal on the third day after the child's admission, and such marked improvement took place that he was dismissed from the hospital on the fifteenth day after admission.

Reexamination on May 11, the day of the patient's dismissal from the clinic and one month after the onset of the infection, revealed the cutaneous lesions to be typically well healed. There was no postherpetic neuralgia. The right eye was

normal as before. Vision of the left eye had improved to 6/6 without correction. The residual moderate thickening of the left upper lid had resulted in a temporarily slightly narrowed palpebral fissure, but there was no ptosis and no evidence of damage to the extraocular motor muscles. The pupil still remained dilated and nonreactive except to mydriatics and miotics. There was some mild residual photophobia. The conjunctival lesions had disappeared, and there was only minimal injection of the bulbar conjunctiva. The cornea was still lacking in sensitivity. It was grossly clear, but examination with the corneal microscope revealed the presence of five small, rounded, faint, grayish, superficial areas of infiltration of the substantia propria in the lower nasal quadrant. Ophthalmoscopic examination gave essentially negative results.

Clinical Notes

TREATMENT OF SERPIGINOUS ULCER OF THE CORNEA WITH METHYL SALICYLATE

S. HANFORD McKEE, M.D., MONTREAL, CANADA

In 1929 Sabatzky [1] claimed to have found in methyl salicylate almost a specific remedy for abrasions, ulcers and opacities of the cornea. His method was as follows:

After anesthetization of the eye with cocaine hydrochloride, the foreign body or unhealthy tissue, as the case might be, was scraped off, and a small drop of methyl salicylate was gently rubbed in with a small glass rod. An ointment containing cocaine was then applied, and the eye was covered. Except for the scraping, the treatment was repeated daily until healing was complete. Sabatzky claimed that for years he has been obtaining practically transparent smooth corneas in cases of corneal foreign body, abrasions and ordinary ulcers, and this induced him to try the method in cases of ulcus serpens. The treatment is the same as that previously described, except that the scraped area is treated with a 1 per cent solution of ethylhydrocupreine hydrochloride before the methyl salicylate is rubbed in. Sabatzky treated 24 patients with pneumococcic ulcers, many of them severe, and reported the method entirely satisfactory. The cure is rapid, there is a minimum of corneal change, and good vision is retained. The beneficial effect of methyl salicylate is attributed to the following factors: 1. It has a germicidal action and a capacity to penetrate deeply into the corneal tissue. 2. It is almost a specific corneal tissue regenerator. 3. Its tension-reducing action on the globe sometimes renders the eye so soft as to give one the impression that the globe had been perforated. 4. It has a capacity to clear up existing scars and also to influence newly-forming corneal lamellae in such a way that from the outset no opaque tissue is formed.

Koldovsky-Kvetoslav [2] reported the use of methyl salicylate according to Sabatzky's method in 2 cases of ulcus rotundum, 4 cases of ulcus serpens, 17 cases of infected foci, 2 cases of herpes and 8 cases of catarrhal ulcer. In no case did the defect heal with a transparent cornea and without the appearance of an opacity. The author infected each eye of 5 rabbits with pus from these patients and treated one eye by Sabatzky's method and the other eye with a solution of ethylhydrocupreine hydrochloride or cautery and found Sabatzky's method to be unsatisfactory, as the ulcers spread beyond control. He stated that there is no justification for employing methyl salicylate in treatment and especially not in cases of ulcus serpens, as the pain is intense and better results may be obtained by other methods.

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1. Sabatzky, K.: Klin. Monatsbl. f. Augenh. **83**:498 (Oct.-Nov.) 1929.
2. Koldovsky-Kvetoslav, abstracted, Am. J. Ophth. **15**:76 (Jan.) 1932.

Since the publication of Sabatzky's article over 20 cases of the serpiginous type of ulcer of the cornea have been treated at the Montreal General Hospital by the use of methyl salicylate as a cautery. In all except 1 of these cases, in most of which the infection was very severe, in some almost desperate, the results have been most satisfactory; in fact, in some cases they can only be described as magical. In practically all cases bacteriologic examination showed numerous lanceolate gram-positive diplococci in the smears, and in many of the cases further bacteriologic examination verified the presence of pneumococci.

The method employed, which differs somewhat from Sabatzky's, follows:

When it was thought advisable, a bacteriologic examination of the ulcerated corneal tissue was made. The eye was anesthetized with a 1 per cent solution of phenacaine hydrochloride. The ulcerated area was thoroughly dried, and the lids were held away from it by the thumb and the first finger. A round-ended wooden applicator was used to apply the methyl salicylate to the ulcer, the surrounding area being kept dry. The oil was rubbed well into the ulcer. After two or three minutes the eye was bandaged, and the patient was put to bed. The ordinary routine tending to raise the body resistance, such as rest and the use of tonics, was carried out. The application of methyl salicylate was repeated three or four times, which in most cases was sufficient. No other form of cauterization was used.

A description of all of these cases would entail considerable repetition, so three or four, typical of them all, will be reported.

REPORT OF CASES

Case 1.—M. M. C., a man aged 50, was seen in the ophthalmic ward of the Montreal General Hospital with a severe serpiginous ulcer of the right eye. The ulcerated area occupied the lower third of the cornea. There was a large hypopyon present. Smears from the ulcerated area demonstrated gram-positive lanceolate diplococci. I first saw the patient during my rounds of the ward with my associates, and the opinion was then expressed that a Saemisch incision was about the only thing that one could do. I had recently read Sabatzky's article and decided to try his treatment. Methyl salicylate was used as described on three different occasions. There was marked improvement following the first application, and after the third the ulcer was entirely healed. The following statements are taken from the house officer's report: "This patient was seen by me in the surgical outdoor clinic four days ago, with a small ulcer of the right cornea, which the patient stated he had had for some days. The ulcer was cauterized with iodine and the patient instructed to return the following day. He was not seen again until three days later, when examination showed a huge deep ulcer, extending over about one-third the surface of the cornea. It looked as if nothing could possibly save his eye. He was sent into the ward, where the ulcer was cauterized with methyl salicylate. This was done three times in all, and the change was almost unbelievable. Following the first application, the ulcer became noticeably smaller and went on to complete cure. The pain he had complained of soon disappeared, as did the hypopyon." The patient was discharged from hospital cured, after a stay of seventeen days.

Case 2.—M. M., a man aged 61, was seen with a serpiginous ulcer of the right eye, occupying the lower third of the cornea. Hypopyon was present. The ulcer

was cauterized with methyl salicylate. Three days later the hypopyon had entirely disappeared, and the eye had improved in every way. The patient recovered completely.

CASE 3.—W. E., a man aged 61, had a large, deep ulcer of the right cornea at the nasal side. A smear from the ulcerated area showed gram-positive lanceolate diplococci. Cauterization with methyl salicylate was followed in three days by marked improvement. The patient was discharged in two weeks, with the ulcer entirely healed.

CASE 4.—H. T. W., a man aged 72, consulted me at my office in July 1937, when he complained of a painful right eye which had been bothering him for a number of days. Examination showed at the temporal side of the right cornea a vertical, deep ulcer, running from the middle third of the cornea to its lower edge. The ulcer seemed so deep and the eye seemed to be in such a precarious condition that no attempt was made to examine the discharge. I felt that the ulcer could perforate at any minute. Both eyes were bandaged, and the patient was sent to the hospital, where the eye was treated with methyl salicylate. His progress was remarkable, so that he was discharged after two weeks. The vision in the right eye was 6/9, with correction. The patient was recently examined for refraction. It was with considerable difficulty that any scarring of the cornea was made out.

While I was putting these notes together, the following was observed:

R. M. B., a man aged 23, was admitted to the ophthalmic service of the Montreal General Hospital after receiving a perforating wound of the right cornea, which had become infected. Examination showed severe blepharospasm and lacrimation, with severe conjunctival chemosis. A vertical laceration extended through the nasal portion of the visual area of the cornea, from 1 to 2 mm. from either limbus. The line of laceration was filled with exudate, and there was much pus in the anterior chamber. The whole cornea was infiltrated and cloudy. A roentgenogram did not reveal any foreign body. The wound was cauterized with methyl salicylate every third day for four treatments. The wound healed, the hypopyon disappeared after the fourth treatment, and the eye became quiet and healed completely.

In 1 case the result was nil, the methyl salicylate having no beneficial effect. The condition was no different clinically from that in the other cases. The patient, a man, had a severe serpiginous ulcer, with hypopyon. Methyl salicylate was applied three times without effect.

All the patients in this series were of middle age or over, except the patient with the traumatic injury. The ulcers were of the serpiginous type; I have not used methyl salicylate in cases of the catarrhal type of ulcer.

In addition to cauterization with methyl salicylate, all of the patients received other necessary treatment, such as rest in bed, tonics and extra feeding. Ethylhydrocupreine hydrochloride was not used in any case.

The purely empiric nature of this therapy is fully realized; nevertheless, I wish strongly to recommend to my colleagues a therapeutic agent that has proved most satisfactory for this destructive form of ulcer. Unquestionably it is a wonderful remedy in certain cases. It avoids the dense scars of the actual cautery; it has not been found painful, as reported by some. Altogether, I recommend it most highly.

Ophthalmologic Review

ECTOPIA LENTIS

A PATHOLOGIC AND CLINICAL STUDY

CLEMENT COBB CLARKE, M.D.
NEW HAVEN, CONN.

In this paper ectopia lentis and congenital dislocated lenses are considered as synonymous terms for describing all lenses dislocated to a greater or lesser extent (luxations and subluxations) on the basis of developmental anomalies. The justification for adding to the already considerable literature on this relatively rare phenomenon rests mainly on the following points: 1. In view of the confusion surrounding the etiology, an anatomic classification of ectopia lentis has been set up which may help to clarify its clinical study. 2. Histologic observations have been presented which illustrate a form of the ciliary body apparently pathognomonic of ectopia lentis. 3. The largest series of cases of ectopia lentis heretofore reported from one hospital has been analyzed in order to emphasize certain clinical aspects and to evaluate surgical therapy for this condition.

DEFINITION

The early writers did not differentiate between the various types of dislocated lenses. According to Celichowska, who has reviewed the early work, some dislocations, judging from the descriptions, undoubtedly were of the congenital type. For example, the earliest case reported (Berryat, 1749) was that of a young man with both lenses in the anterior chamber, likely the end result of congenital subluxation. The distinction between traumatic and spontaneous dislocation, introduced by Sichel in 1846, advanced the understanding of dislocated lenses. The observation of dislocated lenses in children and in persons who had suffered no known trauma or ocular disease led to the concept of a congenital factor producing the luxation (Arlt, 1849). Since the introduction of the term ectopia lentis by Stellwag in 1856 there has been considerable confusion in the use of terms. Ringelhan and Elschnig's comprehensive review in 1931 presented a rational classification of all types of dislocated lenses, which I believe merits adoption. They divided

Based on a thesis submitted to the Faculty of Medicine, Columbia University, for the degree of Doctor of Medical Science in June 1938.

them into (1) traumatic, (2) *consecutiv* or secondary and (3) congenital dislocated lenses. This classification dispenses with the ambiguous term spontaneous dislocation. The traumatic dislocations are obviously the result of injury. The *consecutiv* dislocations are those which are secondary to ocular disease or degeneration, e. g., iridocyclitis, retinal detachment, tumors, infections, chorioretinitis and hypermature cataracts. As Ringelhan and Elschnig said, the only factor in common in this group is the luxated lens. They expressed the belief that ectopia lentis and congenital dislocated lenses were synonymous terms indicating a developmental anomaly in the position of the lens.

ETIOLOGY

The theories as to etiology are numerous and confusing, which is understandable with a condition based on human embryology and occurring so infrequently as ectopia lentis. No one theory serves to explain all cases of ectopia lentis. Most of the theories have been too narrow in their conception, although the influence of heredity has been admitted by all writers in at least a portion of the cases. Reviews [1] of the various theories have been published, notably those by Celichowska, Péré and Ringelhan and Elschnig, so that only a brief summary is included here.

Among the theories regarding the cause of the congenitally dislocated lens are: fluidity of the vitreous; [2] a morbid process occurring in the suspensory ligament similar to that causing cataract (Jäger); absence of the zonule (Klein); incomplete development of the zonule based on intrauterine infection (Sous); rupture of the zonule due to smallness of the lens (Schirmer); malformation of the suspensory ligament resulting from perivasculitis of the small anterior branches of the hyaloid artery and of the vascular capsule of the lens (Vassaux); impeded development of the lens and the suspensory ligament from mesodermal bands and remnants of the hyaloid artery present during the early months of embryonic life but not evident at birth (Hess); atypical direction taken by the invagination of the lens vesicle; [3] stricture of the amnion interfering with the ocular vesicle (Van Duyse), and ectodermal defects resulting from anomalies in the lens anlage itself (Waardenburg). One of the favorite theories is that of Badal and Lagrange, who expressed the belief that the dislocation is due to the anteroposterior elongation of the globe occurring in myopia. The impor-

1. de Caralt; D'Oench; Pires; Pittenger.
2. von Graefe; Desmarres.
3. Helmholtz; Löwe; Samelsohn.

tant Stellwag-Becker theory, which has been supported by Celichowska and others, is that the defect of the zonule occurs as a result of imperfect or delayed closure of the fetal cleft.

Speculation regarding the etiology has been raised by the reports of other congenital anomalies associated with ectopia lentis. Pupillary membrane (Coats) and corectopia [4] are the most frequently associated anomalies. Other associated bulbar anomalies are microphthalmos (case 31), buphthalmos,[5] megalocornea (Thaden), microphakia,[6] coloboma of the lens,[7] coloboma of the iris,[8] and coloboma of the choroid [9] and optic nerve (fig. 1, globe 1158). The recent work on arachnodactylia [10] has brought to light the fact that dislocated lenses and congenital constitu-

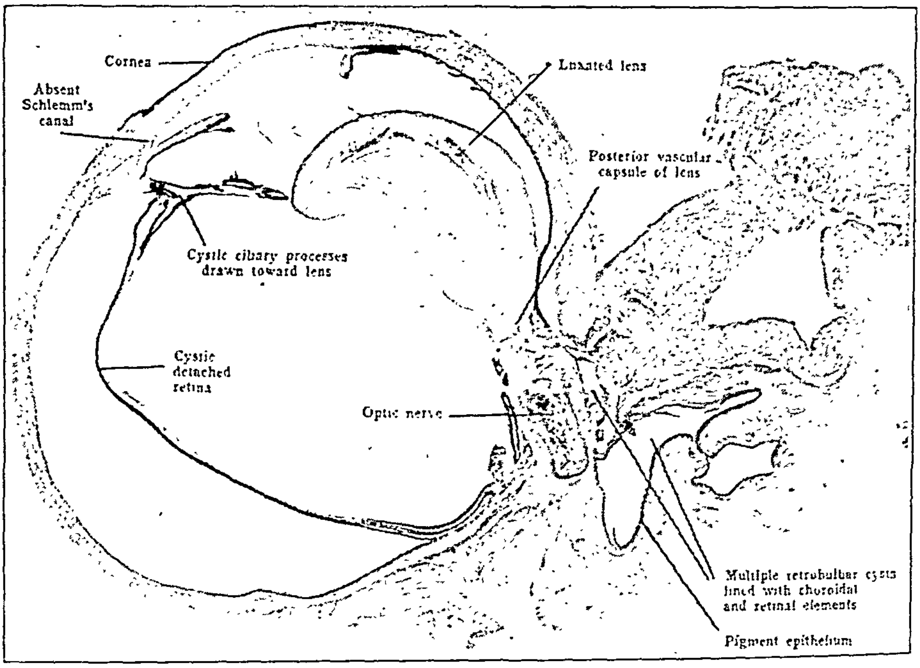

Fig. 1 (globe 1158).—Microphthalmic eye removed from a 10 week old girl. The globe measured 10 by 12 mm. The polycystic mass at the posterior pole shows tissues continuous with the interior of the eye (choroid and retina). The lens is luxated posteriorly by the involvement of its posterior vascular capsule in the cystic mass. The ciliary body on the side opposite to the lens is cystic and drawn backward by fibrous strands. On the side of the lens the ciliary body is nearly absent with a few short ciliary processes; also the angle is rudimentary and Schlemm's canal is missing. The retina also is cystic.

4. de Caralt; Coats; Dixon; Hossmann; Schwarz; Spicer.
5. Allport and Smith; Kurz; Marlow; Péré.
6. D'Oench, 1881; Lindner.
7. Gehrung; Giri; Mann, 1934; Péré; Satanowsky; Weilder.
8. Clark; Guzmann; Kredbova.
9. Addario; Kurz; Wintersteiner.
10. Burch; Isnel and Hakami; Kurz; Lloyd; Morard.

tional defects are more frequently associated than was previously thought. In his excellent review of the Marfan syndrome Burch said that ectopia lentis is found in about one half of the cases of arachnodactylia. Attempts have been made to establish an etiologic relationship between ectopia lentis and these bulbar and constitutional anomalies.

The most recent and perhaps the most authoritative opinion regarding the etiology of ectopia lentis is that of Ida Mann (1937). She regarded the defect as a "dislocation of structures rather than failure." She also said:

> There is no evidence of primary abnormality of any structure but the zonule, in spite of the fact that persistent pupillary membrane and other anomalies of the iris accompany it. [This primary abnormality of the zonule she regarded as an irregular development] with regard to strength and length of fibers, so that when the ciliary ring opens out and should leave the lens suspended in the center of the developing pupil, the lens may show a displacement to one or other side. That this displacement should be more often upwards than in other positions, and very rarely indeed downwards, lends support to this theory, since if the zonule fibres are going to fail they are more likely to do so in the lower part which (being the unstable region of the fissure) is always most likely to show under-development. In such a case the lens would be dragged upwards by the relatively greater pull of the normal fibres at the top. This fits in also with the fact that growth potencies are greatest at the rim of the optic cup at its upper part and its gradients run downwards round the margin towards the fissure.
>
> It is not always justifiable to take refuge in the old theory of persistent mesoderm as an explanation of eye abnormalities. In the case of ectopia lentis we may say that, since it is hereditary (in the same way as certain other admittedly ectodermal abnormalities), it is due to a defect in the tertiary vitreous fibrils at some part of the ciliary ring. It therefore probably begins about the end of the third month, but its potentiality is obviously present from the beginning since it is germinal.

To illustrate the unsettled state as to the causation of ectopia lentis, another recent authority, Duke-Elder, is quoted. In contrast to Mann's opinion that ectopia lentis is due to an ectodermal defect primary in the zonule, he stated:

> The most common cause of an ectopia lentis is a mesodermal aberration; the ætiology of the condition is unknown—presumably it is due to some deleterious chemical influence acting at an early embryonic stage which inhibits mesodermal development, not only in the eye but widely over the body.

In a search for an etiologic basis for ectopia lentis, which by definition is a developmental anomaly, all the factors influencing the development of the embryo must be considered. In a general sense, the anatomic variations from the normal, which are termed congenital anomalies, can be regarded as the resultant of the mutual influence of prenatal environment and hereditary characteristics. The complexities of a study of the etiology of a developmental anomaly are self evident when one even speculates on the countless factors involved in prenatal environment and heredity (Duke-Elder).

HEREDITY

Evidence of the important role played by germinal factors in ectopia lentis has been compiled in Julia Bell's comprehensive review and further confirmed by numerous pedigrees,[11] a partial collection of which was made by Usher. The symmetry and bilateral occurrence of dislocated lenses and the frequency of their occurrence in siblings and in successive generations are strong arguments for the influence of heredity. Pedigrees are on record in which ectopia lentis has appeared in several generations (Pittenger, five generations; Lewis, six generations). According to Mann (1937), "In some families the defect appears almost exclusively in the females; in other families in the males. In others again there is no sex preponderance." The condition may be transmitted by either father or mother to either daughters or sons. While Francheschetti and Díaz-Caneja regarded ectopia lentis as a recessive characteristic because they found consanguinity in families in which ectopia lentis was present, the hereditary element is considered by most authors [12] as being a dominant characteristic, for the defect is most commonly transmitted by a parent who manifests the defect.

It is obvious, as Ringelhan and Elschnig said, that a hereditary factor cannot be shown in all cases of ectopia lentis. Many times lack of investigation or ignorance of the patient regarding his forebears may explain why hereditary transmission is not recorded. On the other hand, investigation in some cases has revealed no known hereditary factor, so that there must be some sporadic cases.[13] Ringelhan and Elschnig cited 1 case in which the parents of an emmetrope with ectopia lentis were found to have myopia but not ectopia lentis. The complexities of heredity are manifold, but its importance in ectopia lentis seems well established.

CLASSIFICATION

An endeavor is made here to set up a classification of the anatomic features of congenital dislocated lenses, which, as previously stated, are the resultant of the interaction of prenatal environment and heredity. Such a classification is obviously artificial when the preliminary stages of the pathogenesis of the condition are not known. Moreover, this classification is one of degree rather than of kind. It proposes to grade the anatomic features found in ectopia lentis from the less severe, involving only the lens supporting structure itself, through the more severe aberrancies, with their associated bulbar anomalies and constitu-

11. Amsler; Cameron; de Caralt; Decker; Díaz-Caneja; Fecht; Francheschetti; A. R. Gunn; Hossmann; Howell; Kredbova; Lewis; Morton; Mules; Pittenger; Roese; von Rötth.

12. Bell; Duke-Elder; Mann, 1937.

13. Bell; Parsons.

tional features. Such a gradation cannot be mutually exclusive, and there is overlapping between the various grades. However, in view of the confusion surrounding the etiology, this classification does seem to clarify the clinical study of a patient with congenital dislocated lenses. The classification is as follows:

I. *Simple Ectopia Lentis.*—This grade is represented by those cases in which the subluxation is due to a defective supporting structure of the lens, i. e., the zonule and the ciliary body. Except for the dislocation of the lens, these eyes are in other respects grossly normal. Slit lamp examinations [14] have sometimes shown the zonular fibers to be attenuated and few in number on the side opposite to the direction of dislocation.[15] Histologic studies have shown the characteristic ciliary body (discussed later) to be present in this grade as well as in the more severe grades of ectopia lentis.

The theories proposed to explain this defective lens-supporting apparatus have been mentioned previously as being either ectodermal (microphakia; abiosis of zonule) or mesodermal (mesodermal bands; hyaloid vessel remains). Since these postulated preliminary steps are regarded as disappearing before birth, the theories remain theories and clinically the only visible result is the dislocated lens.

II. *Ectopia Lentis Combined with Anomalies of Ocular Dimension (Axial Anomalies).*—This grade refers mainly to the combination of simple ectopia lentis with axial myopia, in accordance with the theory of Badal and Lagrange. However, it also refers to any other bulbar anomalies of dimension, regardless of refraction or etiologic theory, so that microphthalmos and buphthalmos are included along with axial myopia as possibly effecting a secondary attenuation of the lens-supporting structures. In regard to myopia, it is generally recognized that most eyes with ectopia lentis are myopic; but not all are myopic due to axial elongation, for the weakened zonule may allow the lens to assume a more spherical shape (Hess; see also the analysis of refraction), thus creating a lenticular myopia, unrelated to the length of the globe. In other cases the myopia may be largely due to a posterior staphyloma which may not affect the anterior segment and the zonule. Thus in this grade are included only those anomalies of the shape or size of the globe which would indicate a probable effect on the suspensory ligament of the lens.

III. *Ectopia Lentis Combined with Anomalies of Ocular Structure.*—This grade refers to those miscellaneous anomalies of bulbar formation which are found more or less frequently in association with ectopia,

14. Bedell; Erggelet; Fox; Hollós; Meesman; Stein.

15. Duke-Elder; Meesman.

even though the possible etiologic relation is not always apparent. These associated anomalies are: pupillary membrane (Crebbin), corectopia;[16] aniridia;[17] polycoria and coloboma of the iris, of the lens or of the choroid;[18] megalocornea (Duke-Elder); retrobulbar cyst (fig. 1, globe 1158), and coloboma of the globe (globe 1158). The wide variety of these associated anomalies includes defects of both ectodermal and mesodermal primitive germ layers, thus confusing the etiologic issue. But when regarded from a clinical standpoint, they represent a convenient though heterogeneous anatomic group of structural aberrancies found in association with ectopia lentis.

IV. *Ectopia Lentis Associated with Constitutional Anomalies.*—Arachnodactylia [19] is the best known constitutional anomaly in grade IV. Other constitutional syndromes less well studied but also associated with ectopia lentis are: dwarfism (Weill), skeletal deformities (Kurz), congenital heart disease (Strebel) and polydactylism (Díaz-Caneja; also case 31 in the present series).

The temptation to correlate ectopia lentis with a defect of a primitive germ layer becomes even greater in dealing with widespread aberrations in body development. According to Mann (1937), this attempt to place the abnormalities in one germ layer "is not at all necessary when we remember the chromosomal linkages between structures of various germ layers already known to occur."

When associated with these constitutional syndromes, ectopia lentis may fall in any of the preceding classifications. It may, for example, be a simple ectopia lentis associated with arachnodactylia, or in another instance it may be ectopia lentis associated with coloboma of the globe, combined with polydactylism. However, for convenience and clarity, this fourth anatomicoclinical gradation has been set for those instances in which constitutional anomalies are associated with the other types of ectopia lentis.

HISTOLOGIC OBSERVATIONS

In the anatomicoclinical classification and later under the topic of clinical aspects (also in table 6) those congenital anomalies associated clinically with ectopia lentis have been considered. In order to show what the relationship of ectopia lentis to other congenital anomalies is from a pathologic point of view, the collection of globes at the Institute of Ophthalmology of the Presbyterian Hospital was reviewed. The collection, accumulated over the eight year period from 1930 through

16. de Caralt; Crebbin; Dixon; Hossmann; Schwarz; Spicer.
17. Batten; Bergmeister; Halben; Neher; Satanowsky.
18. Addario; Gehrung; Giri; Mann, 1934; Péré; Satanowsky; Weilder; Wintersteiner.
19. Burch; Isnel and Hakami; Kurz; Lloyd; Morard.

1937, includes 645 globes, most of which were enucleated at the institute but some of which were sent in from elsewhere for histologic study. Among these 645 globes, 104 showed 26 different types of congenital anomalies (table 1). The incidence of the particular congenital defect, ectopia lentis, among the 104 eyes with any congenital anomaly is 7, or roughly 7 per cent.

In the collection at the institute there were 18 globes with a dislocated lens (table 2). The dislocation was congenital in type in 7, or 39 per cent; dislocations in the others were classified under injuries.

TABLE 1.—*Classification of Twenty-Six Congenital Anomalies Found Histologically in One Hundred and Four Globes*

Anomaly	No. of Globes	Anomaly	No. of Globes
Subluxation of lens	6	Schwalbe's ring	8
Luxation of lens	1	Thinned sclera (blue sclerotics)	1
Vascular capsule of lens	4	Intrascleral nerve loop	22+
Remains of hyaloid artery	12	Retinal folds	2
Pupillary membrane	5	Coloboma of choroid and optic nerve	1
Iris processes	8	Myopia	13
Iris recess	1	Buphthalmos*	13
Coloboma of iris	3	Microphthalmos	1
Iridodialysis and aniridia*	7	Coloboma of globe	1
Ciliary processes on posterior surface of iris	14	Retrobulbar cysts	1
Cysts of ciliary body and processes	10	Cysts of orbit	3
Pigmentation of pectinate ligament	1	Cartilage in orbit	1
Embryonic filtration angle	1	Cyclops	1

* Part of these anomalies were diagnosed as injuries rather than as congenital anomalies.

TABLE 2.—*Types of Dislocated Lenses in Pathologic Collection*

Type of Dislocation	Number
Subluxation, congenital	6
Luxation, congenital	1
Inversion and recession due to injury	1
Luxation in vitreous due to injury	4
Luxation under conjunctiva due to injury	2
Subluxation due to injury	4
	18

The congenital anomalies found associated in the 7 pathologic specimens with ectopia lentis are listed in table 3.

The reasons for enucleating eyes with ectopia lentis are interesting in illustrating the end results in such eyes. As in any condition except a malignant growth, pain or unsightliness is usually the patient's reason for submitting to enucleation. Pain was probably the predominate cause for removal of 4 eyes with glaucoma and of 1 with iridocyclitis, though a staphyloma in 1 eye and buphthalmos in another added a cosmetic reason. Unsightliness was probably the main reason for enucleation of the other 2 eyes, 1 of which was microphthalmic, and contained a large retrobulbar cyst and another of which had bullous keratitis and degen-

eration of the globe. (In table 8 it will be seen that in the present series of clinical cases the 5 eyes which were enucleated all were glaucomatous.) It is probably significant that of the 18 pathologic specimens with all types of dislocated lenses there was an accompanying glaucoma in 5 eyes, of which 4 had the congenital type of dislocation. This might indicate that ectopia lentis progresses to glaucoma, while in the traumatic dislocations the severe disruption of the eye usually necessitates removal before glaucomatous changes have been established histologically.

The incidence of eyes with ectopia lentis seen at the Vanderbilt Clinic has been estimated (table 4) as being 1 : 4,000. The incidence in the pathologic collection at the Institute of Ophthalmology is 7 in 645 globes, or approximately 1 : 100. The seriousness of the condition may be deduced from this comparative incidence: It occurs about forty times more frequently among eyes seriously enough "sick" to be enucleated than it does among eyes affected with the wide variety of ocular conditions seen in the patients of a large ophthalmic clinic.

TABLE 3.—*Congenital Anomalies Associated Histologically with Ectopia Lentis*

Globe No.	Grade	Anomaly
20	II	Subluxation of lens; myopia
818	II	Subluxation of lens; myopia; buphthalmos
948*	I	Subluxation of lens
1013	III	Subluxation of lens; coloboma of iris; buphthalmos
1068*	I	Subluxation of lens; intrascleral nerve loop
1073	I	Subluxation of lens; embryonic filtration angle
1158	IV	Luxation of lens; vascular capsule of lens; coloboma of globe; microphthalmos; retrobulbar cyst; polydactylism

* Globe 948 is from case 25 and globe 1068 from case 12 in the present series of cases.

PATHOGNOMONIC CILIARY BODY AND PROCESSES

As stated in the introduction of this paper, there is a type of ciliary body apparently pathognomonic of ectopia lentis. It may be described as follows:

The ciliary body characteristic of all types of ectopia lentis is quite small with poorly developed or nearly absent circular muscle fibers, and the ciliary processes are extremely underdeveloped and in section appear to point posteriorly. References to the underdeveloped zonule have occurred previously in the literature. For example, Duke-Elder stated: "In cases of simple uncomplicated ectopia the zonule is absent or poorly developed in the direction opposite to the displacement; and it seems that it is the failure of the zonule which is responsible for the deformity." The characteristic ciliary body has also been described by von Hippel, Seefelder and E. Fuchs and is illustrated by A. Fuchs in his "Atlas der Histopathologie des Auges." Pathologic reports have also been made by Terson and Ask. However, Reese in a personal

communication drew attention to the importance of the posteriorly directed ciliary processes. He was the first to emphasize their probable significance as a pathologic diagnostic point.

In an effort to prove or disprove Reese's theory that such posteriorly directed ciliary processes are present in ectopia lentis and in no other condition, 71 of the 645 globes in the collection at the institute were selected for study. These selected globes included most of those which showed the congenital anomalies listed in table 1 as well as those with all types of dislocations (table 2). In 6 of the 7 globes with ectopia lentis the ciliary body was small and the processes directed posteriorly (fig. 2, globe 948). In the seventh globe (fig. 1, globe 1158) the anomalies were so marked (microphthalmos, coloboma of the globe,

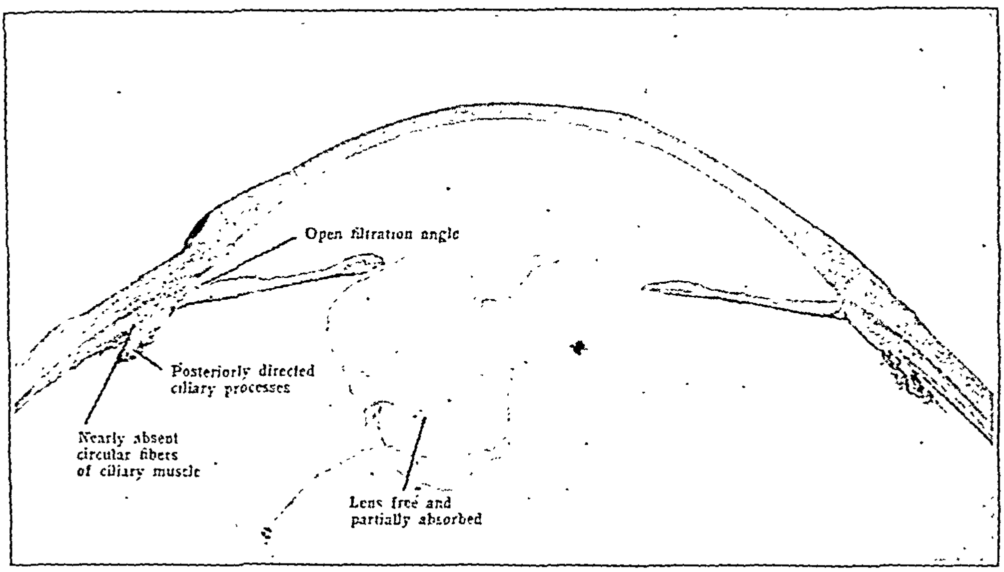

Fig. 2 (globe 948).—Fairly typical example of the ciliary body and processes in ectopia lentis. The nearly absent circular bundles of ciliary muscle and the small and posteriorly directed ciliary processes are noted. The lens, clinically subluxated, is luxated and partially absorbed in the specimen. Glaucoma was present in this eye, although the filtration angle is open.

and retrobulbar cyst) that the ciliary body had no form at all. There was some slight variation in the size and direction of the ciliary body and processes in the 6 globes (globe 1073), but all fitted into the theory. The converse of the theory also was indicated by the anteriorly directed ciliary processes occurring in traumatic dislocations (globe 532), in myopia (in which the ciliary body was sometimes quite undeveloped) and in the other congenital anomalies listed. The exceptions to the rule were found to be seriously injured eyes in which inflammatory and scar tissue adherent to the ciliary processes had obviously dragged the ciliary processes posteriorly (fig 3, globe 383). The photomicrographs demonstrate these statements, and Reese's hypothesis is confirmed.

INCIDENCE

Ectopia lentis is not a common occurrence, yet it cannot be called rare. The figures on the incidence given in the literature vary considerably.[20] The incidence varies mainly with the type of practice: In a general practice, as represented by all patients seen in the ophthalmic clinic of a hospital, the incidence is much less than that in a consultation practice in which the incidence of all types of pathologic conditions is higher. The figure most quoted is that given by D'Oench, who found 10 cases in 50,000 patients at the Knapp Memorial Hospital. Mooren found 20 eyes (11 patients) with ectopia lentis in 157,359 patients; von Becker, 4 eyes in 11,827 patients; Steffan, 11 eyes in 32,595 patients; Arlt, 8 eyes in 5,525 patients; Péré, 5 cases in 6,700 patients; Celi-

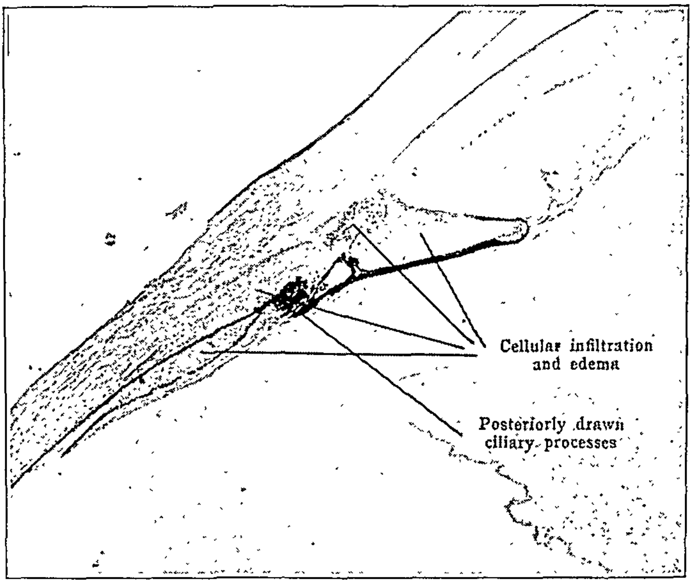

Fig. 3 (globe 383).—Eye with traumatic luxation and detachment of the retina. Inflammatory edema and cellular infiltrate are noted in the filtration angle, iris, scleral spur and ciliary body. The ciliary processes, normal in length, are directed posteriorly due to the traction exerted by the inflammatory process and the detached retina.

chowska 26 cases in 110,000 patients and Ringelhan and Elschnig, 44 eyes in (23 patients) 192,158 patients. Santos Fernández saw only 3 cases in 70,000 patients over a period of forty-three years; Alcalá López observed 2 cases among 8,000 patients in six years, and Miles found 1 case in 10,000 patients.

Of the 31 patients with ectopia lentis in the present series, 11 were seen at the Vanderbilt Clinic over a period of eight years (Jan. 1, 1929 to Jan. 1, 1937), during which time 42,039 new patients were seen.

20. Díaz-Caneja; Dinger; Kurz; Marín Amat; Strebel; Weill.

During the same eight years 18 such patients were seen in Dr. Wheeler's practice; a rough estimate would make the incidence 1:1,000. It is not always clear whether the figures given in the literature refer to eyes or patients. As nearly as these figures can be interpreted, the proportional incidence of cases in the total number of patients is presented (table 4).

TABLE 4.—*Incidence of Ectopia Lentis*

Author	Incidence	Author	Incidence
Alcalá López	1:4,000	Péré	1:1,300
Arlt	1:1,300	Ringelhan and Elschnig	1:8,000
Cellchowska	1:4,000	Santos Fernández	1:23,000
D'Oench	1:5,000	Steffan	1:5,000
Grob	1:7,000	Vanderbilt Clinic	1:4,000
Miles	1:10,000	von Becker	1:5,000
Mooren	1:15,000	Wheeler (estimated)	1:1,000

There is a wide variation in the figures. However, a fair average for an ophthalmic clinic of a hospital would be 1:4,000 or 1:5,000.

POSITION OF LENS IN ECTOPIA LENTIS

The position of the lens in ectopia lentis can be examined from two points of view: (1) the direction of dislocation and (2) the degree of dislocation.

The majority of ectopic lenses are dislocated in an upward direction.[21] Because of the possible relationship of this superior dislocation with the fetal cleft (Stellwag-Becker theory), the direction of dislocation has occasioned considerable study. However, lateral and even downward dislocations have been described,[22] so that too much significance cannot be given to the direction of the dislocated lens, though the opinion of Mann (1937) that "the growth potencies are greatest at the rim of the optic cup at its upper part" and therefore the inferior portion of the zonule is likely to be the weaker may be granted. Another point bearing on the direction of dislocation is the influence of senescence and "subclinical" ocular trauma on a congenitally weak zonule. Thus the patients may be of any age (table 5) when seen, and consequently the original direction of dislocation may have been modified by degeneration or injury of the zonule. By this hypothesis, the older the patient the more likely is the direction of dislocation to be downward and the more likely is symmetry of direction in the two eyes to be lacking (father and two daughters: cases 11, 17 and 18).

The degree of dislocation may vary from all types of subluxation to total luxation. Subluxation has been defined (Ringelhan and

21. Dorsch; Mann, 1937.
22. Duke-Elder; Mann, 1937.

Elschnig) as that condition in which the lens remains partially in the hyaloid fossa but is displaced to one side of the optical axis. Luxation, then, would mean complete displacement from the hyaloid fossa.

Partial dislocation or subluxation may be of any degree. It may even be a potential dislocation which manifests itself at a late age or is indicated only by histologic study of the pathognomonic ciliary body (Reese; also case 25 in the present series). Again, it may be so marked that the edge of the lens is visible in the pupil (fig. 4, case 11), and a phakic and aphakic refraction and view of fundi can be obtained[23] with the possible result of monocular diplopia. Or it may be so slight that a barely perceptible iridodonesis or perhaps a slight inequality of the depth of the anterior chamber or astigmatism unexplained by corneal measurement with a keratometer is the only sign.

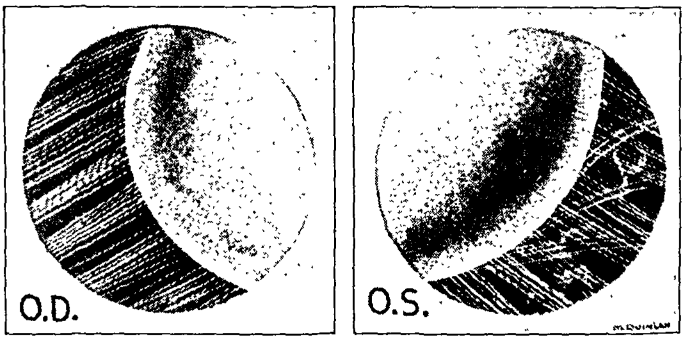

Fig. 4 (case 11).—Ectopia lentis in an Italian married woman aged 27 who had always had poor vision. Her father and two sisters also had ectopia lentis; one brother (not seen) was said to be normal. The drawing shows the upward nasal symmetric subluxation of the lenses as seen through the semidilated pupils. The lenses are clear. The insertion of the attenuated zonule fibers just anterior to the equator may also be noted.

Luxation may be into the vitreous or into the anterior chamber. The zonule may be completely ruptured, or the lens may remain hinged at one point. Such complete displacement from the hyaloid fossa may rarely be the original condition, as when mesodermal bands drag the lens posteriorly into the vitreous.[24] Usually, however, luxation is a result of the transition from a subluxation to complete dislocation occurring either "spontaneously," probably on the basis of senescence or "subclinical" ocular trauma or directly secondary to manifest trauma. In any case the sequelae (especially iridocyclitis and glaucoma) of complete dislocation are manifold and severe.

23. Heddaeus; Leoz; Marquez; Morton; Péré; Williams.

24. Coats; Collins; Kennedy; Seefelder.

CLINICAL ASPECTS

General.—In the usual case of ectopia lentis there are frequently a family history of the condition and bilateral and symmetric subluxation of the lens (fig. 4, case 11). The dislocation is usually upward, but not necessarily so. Phakic refraction usually shows myopia. However, the most important clinical aspect of ectopia lentis is the wide variety of clinical pictures which may be presented. The variability in the direction and the degree of the dislocation has been discussed. The numerous other anomalies, both bulbar and constitutional, which have been shown to accompany ectopia lentis have received consideration in the anatomicoclinical classification previously presented. The frequency of complications in ectopia lentis is another reason for the variable clinical picture. While the emphasis in this paper has been laid on the congenital dislocated lens, it is obvious that in many instances ectopia lentis may be a minor symptom in the individual clinical problem. On the other hand, the detection of an ectopic lens may supply the reason for an acute congestive glaucoma. Again, when corectopia is noted, a realization of its frequent occurrence with ectopia lentis may lead one to detect a barely perceptible iridodonesis.

Diagnosis.—Ordinarily the diagnosis of an ectopic lens is easy. The anterior chamber is deep, the iris and even the lens trembles grossly, the edge of the lens is visible, and the zonular fibers can be seen when the pupil is dilated (fig. 4, case 11). However, when the degree of dislocation is slight, the following points may be of value in establishing the diagnosis. In looking for iridodonesis of slight amount it is better to look midway between the pupil and the periphery for a faint "rippling," similar to that obtained by blowing under a sheet of paper fastened at each end, as the relative rigidity of the pupil and the peripheral attachments are more likely to prevent movement of the inner and outer parts of the iris. Sometimes at examination with the trial case an astigmatism may be found which cannot be explained by the keratometer, or there may be a marked increase of astigmatism from one examination to the next (case 10). Such an event should raise the suspicion of ectopia lentis, although it is not enough to make the diagnosis without corroborative evidence. Another phenomenon which may point toward ectopia lentis is the failure of the pupil to dilate under mydriatics, even atropine [25] (cases 11 and 12). Also, the slit lamp may reveal an anterior chamber of unequal depth which could not be detected grossly, the shallow region being in the direction of dislocation.

Refraction.—The refraction through the lens in cases of ectopia lentis usually shows myopia. It will be shown later in analyzing the cases that the aphakic refraction does not show myopia nearly as fre-

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25. Adam; Cameron; Péré.

quently as has been supposed and that therefore this myopia is sometimes lenticular rather than axial. When the pupil, either with or without mydriasis, is divided by the dislocated lens into phakic and aphakic portions, a "double refraction" may be obtained; that is, correction may be made for the refraction through the lens and through the aphakic portion of the pupil.[23] In the same way a duplicated view of the disk may be obtained with the ophthalmoscope.[26] Likewise there may be monocular diplopia or even bilateral quadrantopia.

Amblyopia.—In ectopia lentis the vision, either with phakic or aphakic correction, is frequently so poor that the question of amblyopia is not considered. However, the presence of a relative amblyopia, especially when operative treatment is under consideration, is of great importance in the prognosis (cases 1 and 7). The large refractive error in ectopia lentis, which often cannot be corrected by glasses, or the constantly changing refraction, due to a movable lens, may play a role in establishing amblyopia ex anopsia. Such an amblyopia may even be bilateral,[27] though it is likely to be more marked in one eye. Then, too, strabismus may be present in ectopia lentis, carrying with it the possibility of amblyopia. Sometimes when the distance vision is equally poor in the two eyes, the patient's preference for the eye used for near vision will point toward an amblyopia.

Strabismus.—Squint, both external and internal, has been observed (Cameron; also cases 5, 11, 22, 23 and 29 in the present series). Nonrefractive squint may occur in cases of ectopia lentis presumably just as it may in other ocular conditions. Refractive squint, it might be supposed, would occur more frequently with this condition, with which anisometropia and myopia are so prevalent. The amblyopia accompanying strabismus has already been discussed.

Complications.—Patients with ectopia lentis have defective eyes; the defect may be limited to a weakened zonule and a potential subluxation, or there may be gross displacement of the lens and other anomalies. The point is that these eyes are more susceptible to injury and inflammation than normal eyes. Luxation of the lens, iridocyclitis [28] and glaucoma are the main complications, affecting not only the visual function but the integrity of the eye. Cataract [29] (cases 6, 16, 19 and 29) and detachment of the retina (Díaz-Caneja; also cases 10 and 20 in the present series) are sometimes found also. Luxation rarely may be present at birth but ordinarily it is the result of a sudden

26. Illustration in Morton (after Streatfield).

27. Giri; Horner; Péré; Raab.

28. Hardy; Hine; Howell; Péré; Terren, Cousin and Kalt.

29. Crebbin; Damianos; Díaz-Caneja; D'Oench, 1881; Fox; Péré; Würdemann.

or gradual change from subluxation. The so-called spontaneous luxation is the probable result of many minor insults to a defective eye. Sudden movements of the head or the eye (what has been referred to as "subclinical" trauma) and the degenerative changes of senescence could affect the weak zonule of the eye with ectopia lentis gradually until the lens became free or nearly free from attachments. Blows on the head or jarring falls ("manifest" trauma) might cause a sudden rupture of the attenuated suspensory ligament of the lens. The events following the conversion into luxation would be determined by the suddenness of the onset and by the direction of the luxation. The totally dislocated lens ordinarily would fall back into the vitreous (cases 2, 3, 10 and 21, right eye), but it could also enter the anterior chamber (Bufill; also cases 16, 18 and 21, left eye, in the present series) or even become strangled in the pupil.[30] The vitreous is sometimes able to tolerate the presence of the lens, but usually a mild or severe inflammation is set up, and glaucoma follows sooner or later. Besides the inflammation secondary to luxation, iridocyclitis often develops when the lens is only subluxated. This form of irritation has been considered as due to the constant striking of a movable lens against the ciliary body (Péré). Glaucoma may be secondary to the inflammation, or it may be set up on a mechanical basis. When the pupil is blocked by the lens and usually when the lens enters the anterior chamber, the intraocular circulation is blocked and glaucoma develops. This mechanical type of glaucoma is likely to be acute, with marked pain and congestion. As for the presence of cataract, opinions vary as to its frequency; Ringelhan and Elschnig found opacities of the lens in 28 of 44 cases, while Damianos found the lenses to be clear in 36 of 40 cases.

Prognosis.—The age of the patient, the severity of the ectopia lentis and the presence and type of associated anomalies are necessary considerations in a prognosis as to visual function and to the integrity of the eye. That the age of the patient is important is obvious, for the possibility of complications is greater the longer the life. That a mild degree of ectopia lentis is no guarantee against complications and even loss of the eye is illustrated by case 25, although the onset was delayed until late in life. The eye with ectopia lentis is a "sick" eye, and as such the prognosis is uniformly grave, though the amount of vision, the duration of useful vision and the time of onset of complications are subject to wide variation,[31]

Treatment.—Therapy for ectopia lentis is directed toward the improvement of vision and the preservation of the globe through the prevention or amelioration of complications. Correction of the refrac-

30. Miles; Ringelham and Elschnig.

31. Celichowska; Foxonet; Pittenger; Putman.

tive errors by glasses is the first step. As has been discussed previously, this correction may be phakic or aphakic, depending on the individual factors. Sometimes the daily use of a mild mydriatic enables a useful portion of the pupil to be exposed (case 13). The patient's regimen must be regulated so that trauma will be reduced to a minimum (de Caralt). Careful, frequent and persistent observation of the patient is necessary to detect and treat complications at their inception.

Operative Treatment.—In operative treatment, too, the primary motive is improvement of visual function. In some cases correction with glasses is insufficient or the lenticular myopia is too high or the position or movability of the lens prevents the formation of a clear retinal image. The general attitude toward operation in such cases of ectopia lentis is one of considerable pessimism.[32] The technical difficulties and the higher incidence of serious surgical sequelae are sufficient explanation for this pessimism, though the surgical-mindedness and dexterity of the ophthalmologist may play a role in his attitude. Certainly one would not rush into an operation on a dislocated lens, but when a low visual acuity justifies operation the added risk should not deter the surgeon from attempting to improve such an important function as vision.[33] Further justification may be found in the frequency and severity of complications, some of which might be forestalled by operation. In view of the variability of the clinical picture in ectopia lentis, the feasibility of operation must, of course, be determined by the factors involved in the individual case.

Types of Operation.—The operations which have been described in the literature fall into two groups: operations on the iris and operations on the lens. Operations on the iris are for the purpose of changing the size or position of the pupil so that diffraction and astigmatism of the dislocated lens may be reduced. Iridodesis,[34] that is, a pulling of the iris to one side as in an iridotasis, is done so as to place the pupil more nearly over the center of the lens. This procedure is now seldom mentioned and was never used much. Sphincterectomy, iridotomy and iridectomy[35] are for the purpose of widening the pupil, so that the center of the lens may be more fully exposed or so that an adequate aphakic portion of the pupil may be used. All operations on the iris are not satisfactory and at best are only palliative, since they do not attack the source of the trouble, i. e., the dislocated lens.

Operations on the lens are for the purpose of removing the lens from the visual axis. The oldest of these is couching. Although this procedure has been practically abandoned for cataract, it is still recom-

32. Brazeau; Leoz; Roese; Terrien.
33. Goldenburg; Weeks.
34. D'Oench, 1881; Péré.
35. D'Oench, 1881; Howell; Péré; Ringelhan and Elschnig.

mended occasionally for the treatment of ectopia lentis. Elliot's monograph on couching would seem authoritative opposition to this procedure, even though occasional cases are seen in which the lens is tolerated in the vitreous (cases 2 and 21, right eye). Discission[36] or capsulotomy is probably recommended more often than any other procedure for ectopia lentis. Since the immediate dangers attending discission are small, it would seem to be the most conservative measure to adopt. However, the objection to discission is its frequent ineffectiveness when one is dealing with subluxated lenses. Some peculiar characteristic of ectopic lenses has been suggested (Reese) as preventing good absorption (fig. 5; case 11, right eye). The more usual explanation is the technical difficulty of getting an adequate opening in the capsule of the movable

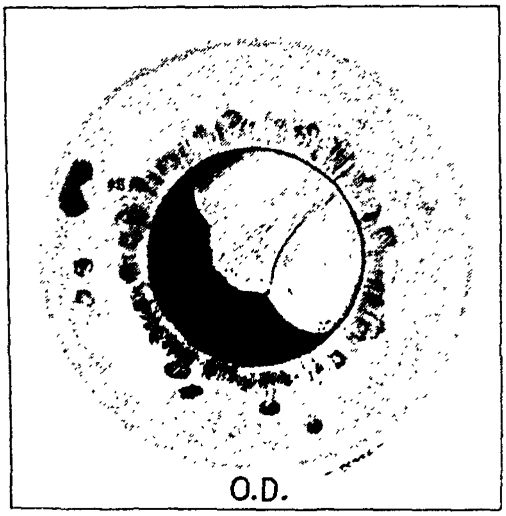

Fig. 5 (case 11).—Incomplete dilation of the thoroughly atropinized pupil (right eye). The poor absorption of the needled lens is clearly shown. Only a small extruded portion of cortex became opaque.

lens. To overcome this, some authors advise the use of two needles, one to fix the lens and the other to tear the capsule.[37]

Extraction is feared by most authors because of the added risk and the technical difficulties. The operative results of extraction reported by Ringelhan and Elschnig and those in the series of cases analyzed here tend to improve the outlook for extraction in cases of ectopia lentis. These results are significant since these two series are larger than any others reported and extraction was employed more often. Ringelhan and Elschnig expressed a preference for intracapsular extraction without iridectomy with the use of the Liegard suture. In the

36. Butler; Decker; Howell; Jackson; Lambert; Morris and Oliver; Péré; Spiece; Strickler; Török and Grout; Weeks; Wray; Zentmayer.

37. Driver; Hine; Horner and Maisler; Parson.

present series the most satisfactory procedure was found to be a wide preliminary iridectomy followed two or more months later by a loop extraction of the lens, conjunctival sutures being employed.

The justification of extraction rests on the fact that the primary cause of the poor vision is removed and the possibilities of future complications inherent in a dislocated lens are diminished. While no universal rule can be laid down for such a variable condition as ectopia lentis, operative intervention is certainly justified in some cases, either to improve vision or to prevent a lens from becoming totally luxated. The added risk of extraction must be balanced against the possible gains, in the calculation of which the presence of amblyopia must be considered. Also, the eventually grave prognosis of the untreated eye with ectopia lentis must be kept in mind.

The operative treatment for the complications is outside the scope of this paper. The treatment for the glaucoma rests with the individual case and the individual surgeon. Luxation of the lens into the anterior chamber bears mention, however, for usually an acute congestive glaucoma results which needs emergency treatment (case 18). Nonoperative treatment in such cases is poor treatment, for it is not effective, and, besides, the presence of the lens in the anterior chamber furnishes an excellent opportunity for its removal. Extraction of a lens from the anterior chamber is relatively easy, if the lens can be made to stay there. Strong miotics to close the pupil behind the lens, fixation of the lens with a needle or extraction with the patient in the prone position (Luedde; also cases 18 and 21 in the present series) are various ways of obtaining this.

DATA FROM THIRTY-ONE CASE HISTORIES

Thirty-one cases from the records of the Presbyterian Hospital are analyzed here. Eleven of the patients entered the hospital from the Vanderbilt Clinic, 18 from the practice of Dr. John M. Wheeler and 1 each from the practices of Dr. John Dunnington and Dr. Hugh McKeown.

The sex ratio is not valuable in such a small series: There were about one and one-half as many males as females.

The ages at which the 31 patients were seen in this hospital are given in table 5. The youngest patient seen was 6 months old; the oldest was 71 years. While many of these patients were seen elsewhere and a diagnosis was made, this table shows a fairly even age distribution of the patients who present themselves for treatment.

A family history of ectopia lentis was obtained for 6 patients, representing three families (cases 11, 16, 17, 18, 26 and 27).

The congenital anomalies found in association with ectopia lentis are recorded in table 6.

Strabismus was present in 5 cases: exotropia and left hypertropia in 1 case, exotropia in 3 cases and esotropia in 1 case. This high incidence of 20 per cent is suggestive, but of course not conclusive in such a small series.

There was glaucoma in 12 eyes of 10 patients, 10 of these being eyes not operated on for improvement of vision. The other 2 eyes with glaucoma were subjected to extractions and were thereby relieved of the glaucoma.

TABLE 5.—*Age Distribution*

Age in Years	No of Patients
1- 4	4
5- 9	4
10-14	3
15-19	1
20-24	3
25-29	3
30-34	3
35-39	0
40-44	0
45-49	2
50-54	2
55-59	3
60-64	1
65-69	1
70-75	1
	31

TABLE 6.—*Congenital Anomalies Associated Clinically with Ectopia Lentis*

Anomalies	Eyes	Patients
Pupillary membrane	7	4
Corectopia	4	2
Heterochromia iridis	1	1
Atrophy of iris	2	1
Poor dilation of pupil	4	2
Coloboma of choroid and iris	1	1
Aniridia and nystagmus	3	2
Cataract	10	6
Coloboma of lens	4	2
Posterior staphyloma	3	3
Microphthalmos	1	1
Polydactylism	..	1

Detachment of the retina occurred in 2 eyes, and in another instance it followed preliminary iridectomy.

Macular degeneration was found in 1 patient.

The anatomicoclinical classification previously presented is applied to this series of cases: grade I, 19 eyes; grade II, 23 eyes; grade III, 12 eyes, and grade IV, 1 eye. Seven were unclassified, owing to insufficient data.

The refraction is known for 35 eyes: The phakic correction was made for 19 eyes, the aphakic correction for 22 eyes and both the aphakic and the phakic correction for 6 eyes. The phakic correction for 17 of the 19 eyes was for myopia; for 1, for emmetropia, and for the other,

for mixed astigmatism. In considering the refraction of the aphakic eyes, all those eyes having a correction of less than +10 D. are considered as having axial myopia, those above +11 D. as being hyperopic, while a few fall into a borderline classification. On this basis, 10 of the 22 aphakic eyes were myopic, 6 were hyperopic and 5 fell into a borderline classification. The greater prevalence of myopia in the phakic correction would corroborate the supposition that a great deal of the myopia in ectopia lentis is on the basis of lenticular myopia. However, the aphakic refractions show that axial myopia is present in about half the cases.

The comparative results of surgical and nonsurgical treatment and of the different types of operations are now considered. In table 7 the number of eyes which were not operated on to improve vision as well as the number which underwent the various types of operations for visual improvement are listed.

TABLE 7.—*Operative Therapy for Present Series of Cases*

Treatment	No. of Eyes
No operation done to improve vision (i. e., needling, preliminary iridectomy or extraction)	41
Needling (2 other needled eyes operated on are listed later)	3
Preliminary iridectomy without extraction	3
Extraction from anterior chamber without iridectomy	2
Extraction with iridectomy	2
Extraction without iridectomy	1
Extraction after iridectomy	10
	62

For the 41 eyes which were not subjected to an operation to improve vision, i. e., preliminary iridectomy, needling or extraction, the possible seriousness of the outcome can be gathered from table 8.

From table 8 it is seen that of the 41 eyes not operated on for visual improvement, glaucoma was present in 10 (5 of which were enucleated), detachment of the retina in 2 and phthisis bulbi in 1. Luxation was present in 9 of these eyes, combined with glaucoma in 6 and with detachment of the retina in 1. Only 2 of the 9 eyes with luxation were useful eyes. The high incidence of glaucoma in cases of ectopia lentis in which operation was not performed is shown, being approximately 25 per cent. Table 8 also shows the high incidence of luxation (about 22 per cent) with its accompanying serious complications.

Of the 21 eyes that were operated on to improve vision, serious results occurred in 3: Hypotony and detachment of the retina developed in 1 after preliminary iridectomy; hemorrhage and prolapse of the iris followed a simple extraction in another; choroidal detachment and iridocyclitis occurred after a combined extraction in the third eye. The details of the various types of operations are given in table 9.

TABLE 8.—*Synopsis of Data on Forty-One Eyes Not Operated on for Visual Improvement*

Case	Age	Eye	Complications	Best Vision*	Operation
2	52	O. D.	Lens in vitreous; posterior staphyloma	20/20	
		O. S.	Glaucoma; luxation in vitreous and anterior chamber	C. F.	Iridectomy for glaucoma
3	27	O. D.	Glaucoma	Nil	Enucleation
		O. S.	Aniridia; nystagmus; glaucoma; luxation in vitreous	C. F.	
4	4+	O. D.	....	20/200	
		O. S.	....	15/200	
7	56	O. D.	Glaucoma	Nil	Enucleation
8	10	O. D.	Amblyopia	20/70	
		O. S.	....	20/40—2	
9	8	O. D.	....	10/200	
		O. S.	....	20/200	
10	53	O. D.	Detachment of retina; luxation in vitreous	H. M.	
11	27	O. S.	Poor dilation of pupil; exotropia......	20/100+1	
12	2	O. D.	Keratitis; glaucoma; poor dilation of pupil	H. M.	Enucleation
		O. S.	Hinged luxation; glaucoma; amblyopia; poor dilation	20/70—1	Trephine with iridectomy
13	46	O. D.	Luxation; glaucoma	Nil	Enucleation
		O. S.	Contracted field	20/40	
15	23	O. D.	Aniridia; nystagmus; luxation; glaucoma	Nil	
		O. S.	Aniridia; nystagmus	C. F.	
16	56	O. D.	Luxation in anterior chamber; cataract; glaucoma; aniridia (?)	H. M.	
		O. S.	Phthisis bulbi	L. P.	
17	12	O. D.	....	20/40+1	
19	71	O. S.	Cataract	20/20	
20	31	O. D.	Heterochromia iridis; posterior staphyloma	20/50	
		O. S.	Detachment of retina..................	L. P.	
21	34	O. D.	Luxation in vitreous; atrophy of iris..	20/15—	
23	24	O. D.	Exotropia	20/40—	
		O. S.	Exotropia	20/30—	
24	18	O. S.	....	20/50	
25	60	O. D.	Glaucoma; keratitis	Nil	Enucleation
		O. S.	....	20/20	
26	7	O. D.	Pupillary membrane	20/100	
		O. S.	Pupillary membrane	20/50	
27	11	O. D.	Coloboma of lens; pupillary membrane	C. F.	
		O. S.	Coloboma of lens; pupillary membrane	20/30+	
28	6	O. D.	....	20/30—1	
		O. S.	....	20/30—1	
29	23	O. S.	Exotropia; corectopia	20/40+	
30	7	O. S.	Coloboma of lens; corectopia; pupillary membrane	20/200	
31	6 mo.	O. D.	Microphthalmos; glaucoma; polydactylism; coloboma of iris; coloboma of choroid and optic nerve	Nil (?)	
		O. S.	....	Normal (?)	

* C. F. indicates ability to count fingers; H. M., perception of hand movements; L. P., perception of light.

From needling, as seen in table 9, only a slight absorption of the lens was obtained. In 2 cases this partial absorption did widen the aphakic portion of the pupil sufficiently to enable an aphakic correction to be used.

From table 10 it is seen that iridectomy in 1 case exposed the pupil sufficiently to give adequate aphakic refraction; vision in 1 eye (case 24, right eye) was lost, though no operative complications furnished an

Table 9.—*Synopsis of Data on Five Eyes in Which Needling Was Done*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
1	O. S.	27	20/50—1 (a)	Unimproved	....	Little absorption
11	O. D.	27	20/200 (p) 20/200+1 (a) when dilated	200/100+1 (a) not dilated	Exotropia	Needled on 2 occasions; enough absorption to expose aphakic part of pupil (fig. 2)
17	O. S.	12	20/100 (p) 20/40 (a) dilated	20/40 (a) undilated	....	Enough absorption to expose aphakic part of pupil
22	O. D.	4	4/200 (p)	Unimproved	Esotropia	Almost no absorption
	O. S.	5	5/200 (p)	Unimproved	Lens attached only above; esotropia	Vitreous tag protruded through wound

* (a) indicates aphakic portion of pupil; (p), phakic portion.

Table 10.—*Synopsis of Data on Three Eyes in Which Preliminary Iridectomy Was Done*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
11	O. D.	27	20/100+1 (a)	20/100+1 (a)	2 preceding needlings; exotropia	Sufficient pupil exposed to give adequate aphakic refraction, but still no improvement in vision; amblyopia (?)
18	O. D.	56	20/200	Unimproved	Incipient glaucoma	Slight loss of vitreous; good recovery
24	O. D.	18	C. F. at 1 foot (30 cm.)	L. P.	....	Hypotony and detachment of retina with no loss of viscid vitreous and reformation of anterior chamber at end of operation

explanation; and in the third case (case 18, right eye) extraction had not yet been done.

The difficulty in the extraction of a lens made accessible by luxation into the anterior chamber is in keeping the lens from falling back. In both of these cases (cases 18 and 21, left eye) the prone position was used to maintain the lens in the anterior chamber. The seriousness of luxation into the anterior chamber is obvious from the poor preoperative vision in both cases and the presence of glaucoma in 1 case and keratitis in the other. Extraction is practically mandatory in such cases and, as indicated by the results in table 11, is justifiable.

It would seem from the 2 cases in table 13 and the 1 in table 12 that incarceration is likely to occur unless the procedure is shortened and the iris controlled by the performance of a preliminary iridectomy.

In this series (table 14) there was loss of vitreous at 5 of 10 preliminary iridectomies and at 7 of 10 extractions after iridectomy.

TABLE 11.—*Synopsis of Data on Two Eyes in Which Extraction Was Done from the Anterior Chamber Without Iridectomy*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
18	O. S.	56	C. F. at 3 inches (7.5 cm.)	20/50+1	Tension, 38	Lens fell back in vitreous with patient supine; restored to anterior chamber by prone position; loss of vitreous; tension became normal after atropine was stopped
21	O. S.	36	C. F. at 2 feet (61 cm.)	20/30+	Keratitis	Patient operated on in semiprone position; slight loss of viscid vitreous

TABLE 12.—*Synopsis of Data on One Eye in Which Extraction was Done Without Iridectomy (Simple Extraction)*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
30	O. D.	8	8/200	Faulty l. p.	Coloboma of lens; corectopia up and out; pupillary membrane	Capsule broke; iris incarcerated; pupil drawn up; iridotomy 1 year later followed by hyphema and secondary membrane; trauma led to hyphema and blood-staining of cornea and faulty projection 1 year later

TABLE 13.—*Synopsis of Data on Two Eyes in Which Extraction Was Done With Iridectomy (Combined Extraction)*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
1	O. D.	26	?	Faulty l. p.	?	Incarceration of iris; good vision immediately after operation; eye became red and painful; choroidal detachment; operation performed in another hospital
29	O. D.	23	C. F. at 3 feet	Unimproved	Exotropia; cataract; corectopia up and out	No loss of vitreous; incarceration of iris; high "hammock" pupil developed which was covered by upper lid

Vitreous was lost from 5 eyes both during the preliminary iridectomy and during the extraction afterward. Yet it will also be noted that incarceration of the iris did not occur. In the 3 eyes in which the vision was unimproved there were no operative or postoperative complications interfering with vision, as indicated by ophthalmoscopic examination

of the media. In 1 of these cases there was macular degeneration; in the other 2 amblyopia may have been the reason for lack of visual improvement. In all the other eyes there was a marked visual gain.

TABLE 14.—*Synopsis of Data on Ten Eyes in Which Extraction was Done After Preliminary Iridectomy*

Case	Eye	Age	Preoperative Vision	Postoperative Vision	Preoperative Complications	Operative and Postoperative Complications
1	O. S.	27	20/50—1 (a)	20/70+ unimproved	Needled previously; small loss of vitreous at iridectomy	Loss of vitreous and hemorrhage into vitreous; discission required; performed without complication
5	O. D.	45	C. F. at 2 feet	20/50—2	Immature cataract; no loss of vitreous at iridectomy; exotropia and left hypertropia	No loss of vitreous
5	O. S.	45	C. F. at 5 feet (152 cm.)	20/20	Immature cataract; exotropia and left hypertropia; no loss of vitreous at iridectomy	No loss of vitreous
6	O. D.	31	4/200	20/30—1	Cataracta caerulea; fluid loss of vitreous at iridectomy	Lens expressed spontaneously; loss of vitreous; vitreous cloudy for a time after operation
6	O. S.	31	20/200	Unimproved	Cataracta caerulea; posterior staphyloma; fluid vitreous at iridectomy	Loss of vitreous; vitreous cloudy for a time; myopic macular degeneration noted after operation when fundus could be adequately seen
7	O. S.	56	20/70	Unimproved	Cataract; vitreous projects into anterior chamber around edge of lens; no loss of vitreous at iridectomy	Small loss of vitreous
10	O. S.	53	6/200	20/70—	Coloboma of lens; some loss of vitreous and small hyphema at iridectomy	Much loss of vitreous; slow recovery; postoperative vision as given was taken only 1 mo. after operation; some opacities of vitreous
14	O. D.	65	1/200	20/40	Nuclear cataract; myopic degeneration of choroid; no loss of vitreous at iridectomy	Slight loss of vitreous
14	O. S.	65	20/200	20/40	Nuclear cataract; myopic degeneration of choroid; no loss of vitreous at iridectomy	No loss of vitreous; rupture of capsule; small hyphema; considerable cortex; discission performed later
19	O. D.	71	4/200	20/30	Senile cataract; loss of fluid vitreous at iridectomy	Loss of vitreous

Of the eyes not operated on for improvement of vision, it will be noted that 22 (53 per cent) are economically blind (20/100 or less). Of the eyes which were operated on for improvement of vision, little or no improvement in vision was obtained after needling or iridectomy (8 eyes), while 9 of 15 eyes subjected to extraction showed improve-

ment of vision postoperatively. Each of 2 eyes showed improvement of vision after extraction of the lens from the anterior chamber, and 7 of 10 eyes showed visual improvement after extraction following preliminary iridectomy, while the results in the 1 case in which simple extraction was done and in the 2 in which combined extractions were done were not so good.

This series, small as it is, would indicate that a more optimistic attitude is justified toward extraction of congenitally dislocated lenses.

SUMMARY AND CONCLUSIONS

A review of the literature, fairly exhaustive since 1920, has been made as well as a study of 71 of the 645 globes in the collection of pathologic specimens of the Institute of Ophthalmology of the Presbyterian Hospital in New York and an analysis of 31 case histories from the records of the Presbyterian Hospital.

Ectopia lentis, synonomously with congenital dislocated lens, is defined as a dislocation of the ocular lens, of greater or less degree, based on a developmental anomaly.

The pathogenesis of ectopia lentis is in a confused state due to many conflicting theories of etiology.

An anatomic classification is proposed to clarify the clinical study of ectopia lentis. It is understood that these gradations are not mutually exclusive, as ordinarily eyes with ectopia lentis will fit into more than one class.

The anatomicoclinical grades are: grade I, simple ectopia lentis; grade II, ectopia lentis combined with anomalies of ocular dimension; grade III, ectopia lentis combined with anomalies of ocular structure; grade IV, ectopia lentis combined with anomalies of constitution, i. e., aberrancies of body development.

Pathognomonic of ecotpia lentis are a poorly developed ciliary body and ciliary processes which are small and point posteriorly, as revealed in histologic specimens.

Amblyopia is frequently present, sometimes in both eyes, and is an important consideration in postoperative prognosis.

There is a high incidence of strabismus.

Phakic refraction nearly always shows myopia (90 per cent). With aphakic refraction, relative myopia is less frequent (45 per cent) but more common than hyperopia (32 per cent) or a borderline condition (23 per cent). This would indicate that a good deal of the myopia in cases of ectopia lentis is lenticular rather than axial.

The ultimate prognosis for untreated eyes with ectopia lentis is bad.

Discissions and operations on the iris are usually unsatisfactory.

Skilful loop extraction following a wide preliminary iridectomy offers the best solution of a difficult problem.

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News and Notes

GENERAL NEWS

Postgraduate Course.—A postgraduate course in ophthalmology will be given at the Center for Continuation Study at the University of Minnesota, Jan. 16 to 21, 1939. The course is recommended primarily for ophthalmologists, but all physicians are invited to attend. Physicians residing outside the state are accepted on the same basis as those living in Minnesota. The subject matter will be presented in the form of illustrated lectures, movies, demonstrations and round table question and answer periods. Clinics will not be attempted because of the difficulty in showing patients properly to more than a few physicians at a time. The regular faculty will be augmented by the following guest speakers: Dr. William Thornwall Davis, Washington, D. C.; Dr. Sanford Gifford, Chicago; Dr. Harry S. Gradle, Chicago, and Dr. Albert D. Ruedemann, Cleveland.

The fee for the course is $25. Further information can be obtained from Dr. William A. O'Brien, director of postgraduate medical education, University of Minnesota, Minneapolis, Minn.

Course in Ophthalmology, Harvard University Medical School.—Harvard University Medical School offers the following courses in ophthalmology for the first half of 1939:

From February 6 to 18 a course on ocular muscles will be given. This course includes the neuroanatomy and physiology of the ocular muscles as an introduction to didactic and clinical work and deals extensively with vertical deviations. It does not include orthoptics. The course is given by Drs. Bielschowsky and Casten.

From March 1 to 31 a course on the use of the slit lamp will be given by Dr. Beetham; a course on external diseases of the eye by Dr. Gundersen, and a course on ocular complications in general disease by Dr. King. These courses may be taken simultaneously.

From April 10 to May 6 a course in recent advances in ophthalmology will be given. This course correlates the pathology of the eye and clinical practice. It draws on all resources of the ophthalmic department of the Massachusetts Eye and Ear Infirmary, clinical, laboratory and research. Although this course has pathology as a basis, it gives a cross section of all work at the infirmary. The section on pathology is given by Dr. Terry.

In July a course on visual optics and physiology will be given by Drs. Ludvigh, Cogan and Easton.

Beginners and general practitioners may take the course in ocular complications in general disease. All other courses listed are not open to beginners. Further information may be obtained from the assistant dean of the medical School.

Annual Course, Research Study Club of Los Angeles.—The eighth annual midwinter clinical course will be given by the Research Study

Club of Los Angeles from January 16 to 27 inclusive. It will embrace both ophthalmology and otolaryngology.

The teaching staff will include: Prof. Gösta Dohlman, professor of otolaryngology, Lund University, Lund, Sweden; Dr. Edward Jackson, emeritus professor of ophthalmology, University of Colorado Medical School, Denver; Dr. John F. Barnhill, emeritus professor of head and neck surgery, University of Indiana, Indianapolis; Dr. William L. Benedict, head of the department and professor of ophthalmology of the Mayo Foundation, Rochester, Minn.; Dr. Phillips Thygeson, assistant professor of ophthalmology of the College of Physicians and Surgeons, Columbia University, New York; Dr. George N. Hosford, ophthalmologist to the Children's Hospital, San Francisco; Dr. Augustus Pohlman, former professor of anatomy, Creighton University Medical School, Omaha; Dr. Simon Jesberg, assistant professor of otolaryngology, University of Southern California, Los Angeles; Dr. Louis K. Guggenheim, assistant professor of otolaryngology, Washington University, St. Louis; research associate, University of California, Los Angeles; director of research in otolaryngology for the Research Study Club of Los Angeles.

Instruction courses in ophthalmology will be given as follows: "Diseases and Disorders of the Orbit: Treatment and Surgery," by Dr. William L. Benedict; "Procedures of Value in the Diagnosis and Management of Ocular Diseases," by Dr. Phillips Thygeson; "Hydrogen Ion Phenomena in Relation to Ophthalmology," by Dr. George N. Hosford; "Practical Methods of Refraction," by Dr. Edward Jackson, and "The Pathology of the Uveal Tract," by Dr. M. N. Beigelman.

American Board of Ophthalmology.—The American Board of Ophthalmology announces an important change in its method of examination of candidates for the board's certificate.

Examinations will be divided into two parts. Candidates whose applications are accepted will be required to pass a written examination, which will be held simultaneously in various cities throughout the country approximately sixty days prior to the date of the oral examination.

The written examination will include all of the subjects previously covered by the practical and oral examinations.

Oral examinations will be held at the time and place of the meeting of the American Medical Association and of the American Academy of Ophthalmology and Otolaryngology and occasionally in connection with other important medical meetings. The oral examination will be on the following subjects: external diseases, ophthalmoscopy, pathology, refraction, ocular motility and practical surgery.

Only those candidates who pass the written examination and who have presented satisfactory case reports will be permitted to appear for the oral examination.

Examinations scheduled for 1939 are as follows: written examinations, March 15 and August 5; oral examination, May 15 in St. Louis and October 6 in Chicago.

Applications for permission to take the written examination on March 15 must be filed with the secretary not later than February 15.

Application forms and detailed information should be secured at once from the secretary, Dr. John Green, 6830 Waterman Avenue, St. Louis.

Anniversary Number of Annales d'oculistique.—The January 1938 number of the *Annales d'oculistique* is devoted to a celebration of the hundredth anniversary of the journal. The *Annales* was originally a Belgian publication, founded by Cunier in 1838, and has always been conducted by a French and Belgian editorial board.

The foreword written by Cunier for the first number of this periodical in 1838 and the table of contents of the first volume are reissued. Then follows an article by Rochon-Duvigneaud, in which he reviews one hundred years of French and Belgian ophthalmology, with seventeen excellent photographs of outstanding French and Belgian ophthalmologists. Some old papers which are characteristic of their eminent authors are reprinted: "Paralysis of the Associated Ocular Movements," by Parinaud in 1883; "Sclerotomy," by Lagrange in 1906; "Vision," by Nuel, with an analysis of the article by Parinaud in 1904; "Gonorrheal Infections," by Morax, and "Laqueur's Discovery of the Antiglaucomatous Action of Eserin," by Redslob in 1876.

The editorial board of the present *Annales* consists of Magitot, chief editor; Rochon-Duvigneaud, Bailliart, Hartmann, Leplat, Dupuy-Dutemps, Redslob, Jeandelize and Hambresin.

Theodore Axenfeld Prize.—The publishing firm, Ferdinand Enke Verlag, in Stuttgart, Germany, has decided to donate a Theodore Axenfeld Prize. This prize of 500 reichsmarks will be distributed every two years to the most meritorious article which has appeared in the *Klinische Monatsblätter für Augenheilkunde.* The selection will be made by five members: the chief editor, the president of the German Ophthalmological Society and three members, who will be selected by the editor and publisher.

The winner of the prize is to be announced at the meeting of the German Ophthalmological Society.

Trachoma Research.—The Indian Medical Service has set up a research program in trachoma under the supervision of Dr. Polk Richards and Dr. Fred Loe, of the Indian Medical Service, and Dr. Phillips Thygeson, of the Eye Institute, New York. The committee will carry on research treatment of trachoma with sulfanilamide and sulfanilamide derivatives. A comparative study of therapy with sulfanilamide and antimony and potassium tartrate will be made by Dr. Julianelle, of St. Louis, in cooperation with the physicians in the Indian Medical Service.

SOCIETY NEWS

American Academy of Ophthalmology and Otolaryngology.—Dr. Albert C. Snell, Rochester, N. Y., of the University of Rochester School of Medicine, was named president-elect of the American Academy of Ophthalmology and Otolaryngology at its forty-third annual session in Washington, D. C., October 9 to 14.

Dr. George M. Coates, Philadelphia, took office as president, having been elected at last year's meeting in Chicago.

Three vice presidents were elected: Drs. William W. Pearson, Des Moines, Iowa; William J. Mellinger, Santa Barbara, Calif., and Charles A. Bahn, New Orleans. Dr. William P. Wherry, Omaha, was reelected executive secretary-treasurer, and Dr. Secord H. Large, Cleveland, comptroller. Dr. Carl H. McCaskey, Indianapolis, was elected a member of the council, and Dr. Grady E. Clay, Atlanta, Ga., was chosen to represent the academy on the American Board of Ophthalmology.

The 1939 meeting will be held in Chicago.

Canadian Ophthalmological Society.—The Canadian Ophthalmological Society held its first annual meeting in Montreal, Aug. 24 to 25, 1938. Sir Stewart Duke-Elder, lecturer in ophthalmology, of St. George's Hospital Medical School, University of London, was the guest of honor, speaking on "Progress in Ophthalmology." A symposium on visual standards was presented, and a committee was appointed to formulate standards which will be available as an authoritative expression of the society. The second day was spent in clinical sessions at the Royal Victoria Hospital. The following officers were elected: Drs. W. Gordon M. Byers, Montreal, president; William H. Lowry, Toronto, vice president, and Alexander E. MacDonald, Toronto, secretary.

International College of Surgeons.—The International College of Surgeons will hold its assembly in New York at the Hotel Roosevelt on May 22-24, 1939. Dr. Edward Frankel Jr., 217 East Seventeenth Street, New York, has been appointed by the officers of the college as general chairman of this assembly. Any one interested in space for scientific exhibits may communicate with him.

Information, Statistics, Photographs Wanted for Book Concerning Industrial Eye Hazards.—The National Society for the Prevention of Blindness has issued a public call for (1) information concerning new industrial or occupational hazards to eyes, including both accident and disease hazards; (2) recent and significant statistics concerning any occupational hazards to sight, showing frequency, severity, causes, nature of injury, degree of impairment and cost; (3) photographs showing either hazards to sight or protection against such hazards, and, most important of all, (4) information concerning successful methods of eliminating, counteracting or alleviating the disease and accident hazards to eyes. The society's headquarters are at 50 West Fiftieth Street, New York.

This information is desired for consideration in the revision of Eye Hazards in Industrial Occupations by Lewis H. Carris, managing director of the society, and Louis Resnick.

De Schweinitz Lecture.—A lecture in honor of Dr. George E. de Schweinitz has been founded by the Section on Ophthalmology of the College of Physicians of Philadelphia, to be known as the de Schweinitz Lecture. The lecture is to be given annually on the third Thursday of November, on a subject to be chosen by the speaker selected.

S. Lewis Ziegler Prize.—Through the generosity of Mrs. Kathryn Ziegler Halsey and S. Lewis Ziegler Jr., the S. Lewis Ziegler Prize has been established by the Section on Ophthalmology of the College of Physicians of Philadelphia. One hundred dollars is annually offered for an original contribution to ophthalmology deemed of sufficient merit, the committee of award to be appointed by the chairman of the section. Theses for the 1938-1939 award must be in the hands of the committee by Oct. 1, 1939.

Iowa Academy of Ophthalmology and Oto-Laryngology.—Dr. J. K. Von Lackum, of Cedar Rapids, was elected president of the Iowa Academy of Ophthalmology and Oto-Laryngology for the 1939-1940 term at the annual convention of the organization in Davenport, Nov. 14, 1938. Dr. Dean M. Lierle, of Iowa City, succeeded Dr. Harry H. Lamb of Davenport as president for the coming year. Dr. Byron M. Merkel, of Des Moines, was reelected secretary-treasurer.

It was decided that the 1940 meeting will be held in Cedar Rapids. It had previously been decided that the academy will convene in Iowa City in 1939.

UNIVERSITY NEWS

Wayne University.—A total of $45,000 has been accepted by the Wayne University College of Medicine, Detroit, in support of research and teaching at the College of Medicine. The largest single gift was $10,000 for the establishment of a research laboratory in ophthalmology. Dr. Parker Heath, professor of ophthalmology, has charge of the laboratory. Gordon L. Walls, Sc.D., has been appointed research associate in ophthalmology.

PERSONAL

Dr. William B. Clark, of New Orleans, was recently made associate professor of ophthalmology at Tulane University School of Medicine, New Orleans, and placed in charge of the ophthalmic department of the Hutchinson Memorial Clinic and of graduate instruction in ophthalmology at the Eye, Ear, Nose and Throat Hospital.

PERSONAL

Dr. J. Warren Bell has been appointed medical director of the National Society for the Prevention of Blindness.

CORRECTION

In the article by Dr. Edmund B. Spaeth entitled "Etiology of Retinal Separation Considered from the Standpoint of Surgical Correction," in the December issue (Arch. Ophth. **20**:1046, 1938), the date "1893" in the twelfth line on page 1047 should read "1853."

Correspondence

PROLAPSE OF THE IRIS AT CATARACT OPERATION

To the Editor:—I have read Dr. Frank C. Parker's paper on a cataract operation to reduce the incidence of prolapse of the iris and his instruction on the movements of the iris during the incision with great interest (ARCH. OPHTH. 20: 597 [Oct.] 1938). Like him, I have had an occasional prolapse after simple extraction of a cataract, but I adopted the small peripheral iridectomy to allow the fluid behind the iris to escape through the wound without prolapse of the iris. Since adopting this simple procedure many years ago, I have scarcely ever had a prolapse.

The only difficulty was that when iridectomy was done first the lens attempted to come through the coloboma instead of through the pupil. For some time I have done the peripheral iridectomy after the extraction, and this has been entirely satisfactory.

One drop of physostigmine salicylate after the operation insures firm contraction of the sphincter pupillae and can be followed by the use of atropine at the first dressing the next morning.

W. B. INGLIS POLLOCK, M.D., GLASGOW, SCOTLAND.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

TOPOGRAPHY OF MEDULLARY IRIDODILATOR FIBERS. J. DECHAUME and G. MORIN, Compt. rend. Soc. de biol. **127:** 1486, 1938.

The experiments of the authors demonstrate that in the dog no iridodilator fibers leave the cord via the fifth, sixth or seventh cervical roots. In animals aseptically deprived of their spinal medulla below this region, and with the adrenal glands cut off from neural influence, the Horner syndrome resulted and the pupil failed to respond by dilatation to emotional stimuli. The effect was limited to the side destroyed, indicating that in the dog the sympathetic innervation to the eye is entirely homolateral.

J. E. LEBENSOHN.

Congenital Anomalies

CONGENITAL TORSIONAL NYSTAGMUS. J. OHM, Arch. f. Ophth. **138:** 693 (May) 1938.

Seventy cases of congenital torsional nystagmus are thoroughly analyzed. Ohm's method of lever nystagmography is not suitable for the recording of pure torsion. The average visual acuity in cases of congenital torsional nystagmus is definitely better than in cases of albinism or of colobomas of the uvea. Characteristic of congenital torsional nystagmus is relatively good vision (better than 0.5; average, 0.68) in one eye combined with relatively poor vision (average, 0.25) in the other eye. Other motor anomalies are common in cases of this condition. Strabismus occurs in 61.4 per cent; in approximately half of these cases a constant horizontal deviation is combined with a vertical component the direction and degree of which varies in the different parts of the field of fixation (oblique strabismus). The mechanism of this form of strabismus is made the subject of lengthy speculative interpretations which do not lend themselves to abstracting.

P. C. KRONFELD.

Experimental Pathology

OBSTRUCTION OF THE IRIDOCORNEAL ANGLE BY NEGATIVE PRESSURE. F. KUKAN, Klin. Monatsbl. f. Augenh. **100:** 68 (Dec.) 1938.

Kukan conducted a series of experiments on 15 patients to find out the cause of the decrease of intraocular tension after compression of the eyeball by massage or by the application of a tonometer or an ophthalmodynamometer. This phenomenon occurs, in his opinion, as the result of elimination of the aqueous humor through the iridocorneal

angle. He describes his method of examination by means of an ophthalmodynamometer and a small vacuum cup, which, placed over the cornea, rests on the limbus. A negative pressure of 150 mm. was produced by so-called pericorneal suction lasting three minutes, after which the tension was taken by the tonometer. He found that pericorneal suction failed to lower the tension and that it raised it in 1 case. So-called scleral suction, on the other hand, done with a larger cup produced a decided decrease in the tension of the other eye in this case. In those cases in which only a slight change in intraocular tension, or none at all, was obtained by pericorneal suction, no change was observed immediately after scleral suction. Expression of the blood during suction is irrelevant in lowering the tension, because the blood will return into the blood vessels as soon as the cup is removed.

The stretching of the sclera and the reduction of its elasticity occurs more distinctly during pericorneal, than during scleral, suction. Hence, the reduction of the tension resulting from stretching of the sclera is greater with pericorneal suction. This observation suggests that the difference in lowering the tension by these two methods is not increased but actually decreased by stretching and loss of elasticity of the sclera. Kukan concludes that in increased intraocular tension produced by compression the iridocorneal angle is the main passage for elimination of the aqueous humor.

K. L. Stoll.

General Diseases

Leprosy in Ophthalmology. Viallefont and Fuentes, Ann. d'ocul. **175:** 380 (May) 1938.

The role of leprosy as a factor in blindness is important. Though the disease is rare in France, it is fairly common in certain quarters of the world. Of 31 patients examined, 22 presented more or less important lesions and 9 were blind. Nearly all the patients had definite iritis, with exudation and numerous synechiae. In 1 the iritis took on a peculiar aspect, such as that to which Cange has drawn attention. In a large number of the patients different forms of keratitis were present, either as a diffuse leukoma or as isolated, round, grayish lesions. The authors did not note any appreciable lesions of the fundus in those cases in which they were able to examine the interior of the eyes. They did not observe atrophy of the optic nerve or chorioretinitis, but the examination was rendered impossible in many instances by the lesions of the anterior segment.

From a clinical point of view, the lesions that the authors observed were similar to those of tuberculosis. The differential clinical diagnosis between tuberculosis and ocular leprosy is difficult, but when leprosy is evident, laboratory examination simplifies the matter.

S. H. McKee.

Constitution and Ocular Tuberculosis. W. Steinberg, Klin. Monatsbl. f. Augenh. **99:** 784 (Dec.) 1937.

Steinberg examined 100 men and 100 women in whom the tuberculous genesis of their ocular disease was either firmly or, at least,

convincingly established. Children and young adults were excluded, because their type of constitution cannot be determined definitely. The physical constitution of these patients was determined by Kretschmar's method, which includes the collection of anamnestic, somatometric and descriptive data. The various types of constitution are described and recorded in tables. Steinberg could not corroborate the findings of Brückner and of Gigon, which were based on examination of natives of Switzerland. Steinberg's patients with ocular tuberculosis were not of a predominantly pyknic type, but they were fairly evenly distributed in the three types, the pyknic, the athletic and the leptosomatic. The difference of Brückner's findings may be due to a greater admixture of the Eastern and Alpine races than in Steinberg's territory, the Black Forest, and among his other patients who came from cities and country districts in Germany. Numerous dysplasias, especially those of a hypogenital type, and frequent thyroid anomalies found in his patients, prompt Steinberg to suspect a partial constitutional anomaly favoring atypical localization of ocular tuberculosis. This supposition receives support by the almost general benignity of the pulmonary tuberculosis in the patients with ocular tuberculosis. Their robust build and good nutritional condition would suggest a lymphatic condition resembling a hypoplastic status, in which the lymphatic condition has to be considered as a partial manifestation. Frequent attacks of catarrh of the upper part of the respiratory tract and nasal adnexa, scrofulosis, abscesses of the glands and rheumatism of the joints in these patients may be explained in this connection. Atypical localization of the tuberculosis of these patients in the eye is probably due to inferiority of the ocular tissues, as a result of general constitutional anomaly, brought on by their lymphatic or hypoplastic status. Decreased resistance of the eye, on the other hand, may be caused by previous traumatic lesions or nontraumatic diseases.

K. L. Stoll.

Glaucoma

New Developments in the Pharmacological Treatment of Primary Glaucoma. M. J. Schoenberg, Brit. J. Ophth. 22: 417 (July) 1938.

Schoenberg reviews the essentials of the theory of chemical mediation of nerve impulses, the mode of action of sympathicomimetics (Sy-Mi) and parasympathicomimetics (Pa-Sy-Mi) and the rationale of the pharmacologic local treatment of glaucoma.

The author gives the following summary:

"1. Nerve impulses travelling along the two branches of the vegetative nervous system are transmitted to the secretory or contractile cells by the aid of a chemical mediator (acetylcholine or sympathin, respectively).

"2. There are evidences which support the assumption that in primary glaucoma, there is a disturbance (functional or organic) of one or both branches of the vegetative nervous system, within the eye.

"3. The present pharmacological treatment of primary glaucoma endeavors to correct such a disturbance by dealing with each or with all three of the components of the neuro-effector unit.

"4. As far as we know, drugs that have a beneficial effect upon eyes with a primary glaucoma, possess the property of acting either on the esterase (which neutralises acetylcholine) or on the effector cells.

"5. There is need of more knowledge on the subject of dysfunction or degeneration of the parasympathetic and sympathetic nerve terminals, more information on details regarding the process of release of acetylcholine and esterase, also on affectations of effector cells. There is also need of drugs or other means which can re-establish the normal function of any one of the three components of the neuro-effector unit, whenever impaired.

"6. Clinical experience leads us to believe that in some cases of primary glaucoma, we are dealing with a failing function of the ocular parasympathetic, in others with a preponderance of the ocular sympathetic, and in still other cases with a combination of both conditions. Thus, we may classify—for the present—the various types of primary glaucoma in three groups: sympathetic, parasympathetic and mixed.

"7. The adoption of this classification may prove to be helpful to the progress of our knowledge of the pharmacological treatment of primary glaucoma."

W. ZENTMAYER.

Injuries

WHY OPERATE EARLY IN CASES OF SEVERE BURNS OF THE EYE? O. THIES, Arch. f. Ophth. 138: 686 (May) 1938.

For more than thirty years Denig has been advocating his method of covering severely burned areas of bulbar conjunctiva with free transplants of mucous membrane shortly after the injury. Thies, the author of a recent monograph on "Burns of the Eye" (Stuttgart, Ferdinand Enke), now summarizes the experimental and clinical evidence which made him adopt Denig's method. The operation should be performed within the first seven hours after the injury, but "not much is lost if one performs the operation within the first twenty-four hours." The evidence presented in the paper appears to show that the results of early operation are definitely better than those of conservative treatment. The mechanism of this beneficial influence of the surgical treatment is not clear.

P. C. KRONFELD.

Instruments

MEASUREMENT OF THE ANGLE OF MAXIMUM CONVERGENCE. H. G. MARTIN, Am. J. Ophth. 21: 161 (Feb.) 1938.

Martin describes a method for measuring maximum convergence which he considers a more representative and equitable means of determining this function than other methods. The results lend themselves more readily to comparison with the accommodative function. The instrument used is described.

W. S. REESE.

A New Ophthalmodynamometer. J. Keil, Klin. Monatsbl. f. Augenh. **99:** 625 (Nov.) 1937.

The use and scope of the ophthalmodynamometers are described, and the advantages of Keil's own device are reported in detail. This instrument is a compression dynamometer, containing no springs. The values obtained with it are reliable and accurate; they may be read easily on scales on both sides of the apparatus. Owing to its symmetric construction, this apparatus may be used for both eyes; it is small and easy to handle.

K. L. Stoll.

Lens

Vitamin C Balance in Patients with Senile Cataract. J. Seefried, Arch. f. Ophth. **138:** 620 (May) 1938.

The relation between vitamin C intake and excretion in patients with senile cataract was determined by means of the tolerance test of Jezler, Kapp and Niederberger. The patient receives daily, in addition to his ordinary diet, 300 mg. of vitamin C by mouth. This abnormally high intake raises the vitamin excretion in the urine. The number of days which must elapse until the patient's system becomes so saturated with vitamin that 50 per cent of the daily intake is excreted again in one day has been found to be a significant index of the patient's vitamin C metabolism. Comparing 44 patients with senile cataracts with 25 controls of approximately the same age, the author found that, on the average, it takes the patient with senile cataract three days longer than the control to reach the point where 50 per cent of the vitamin intake is excreted again. This suggests the presence of a general disturbance of the vitamin C metabolism in cataractous patients.

P. C. Kronfeld.

Experimental Studies on Roentgen Cataract. H. Goldmann and A. Liechti, Arch. f. Ophth. **138:** 722 (May) 1938.

The authors solved the problem of "focusing" roentgen rays on certain portions of the lens by using a very narrow, but concentrated beam and by determining its position with regard to the eye by dusting a fluorescent powder on the cornea. The distance between the focus of the tube and the cornea was 29 cm., and the voltage was 200 kilovolts. The radiation was filtered through 1 mm. of aluminum. The doses applied to the surface of the cornea varied from 1,500 to 2,000 roentgens. At a distance of 15 mm. from the irradiated surface, about 90 per cent of the surface doses were still present. With this technic the authors irradiated in rabbits (*a*) peripheral portions of iris and lens, (*b*) only axial portions of lens and (*c*) only peripheral portions of lens (several months after iridectomy). In the animals of the first group opacities of the lens developed about in from three to four months after irradiation. The first opacities became visible in axial portions of the lens, near the sutures, that is, in portions which had not been irradiated. No opacities of any kind developed in the animals in the second group. The axial portions of the lens thus proved to be insensitive

to direct irradiation. In the animals belonging to the third group axial opacities of the lens were noticed, similar to those found in the first group. The authors conclude that the "point of attack" (*Angriffspunkt*) of the roentgen rays is the equator of the lens. The iris and the ciliary body play no demonstrable part in the development of roentgen cataract. The first visible changes occur at the extremities of the lens fibers. In the region of the opacities one can observe the formation of a new suture, which again indicates damage to the germinative lens epithelium that manifests itself as formation of shorter fibers.

P. C. KRONFELD.

Methods of Examination

ENTOPTIC DEMONSTRATION OF THE YELLOW COLOR OF THE MACULA CENTRALIS RETINAE. R. ZUBLER, Arch. f. Ophth. **138:** 633 (May) 1938.

In 1925 A. Vogt reported that the yellow color of the macula can be made visible entoptically by diascleral incidence of red-free light rays emitted by an arc lamp which is run on direct current. The author of the paper under review, under Vogt's guidance, has made a thorough study of the conditions governing this entoptic phenomenon and of its parallactic modifications. No new facts are reported, but previous observations are confirmed and thoroughly analyzed.

P. C. KRONFELD.

Neurology

HYPERTROPHIC INTERSTITIAL NEURITIS WITH PAPILLEDEMA. A. W. DIDDLE and R. L. STEPHENS, Arch. Neurol. & Psychiat. **40:** 1 (July) 1938.

A case of hypertrophic interstitial neuritis, a condition first described by Dejerine and Sottas in 1893, is presented. The patient, a 26 year old man, gave a history of pain, numbness and weakness of arms and legs with increased difficulty in walking for two years and intermittent blurring of vision for three years.

The principal clinical findings of significance were: hypesthesia of the glove and stocking type; partial paralysis and muscular atrophy of the extremities; hypertrophy of the peripheral nerves and moderate loss of vision. Associated with this were bilateral papilledema and increase in the cerebrospinal fluid pressure and the total protein content of the spinal fluid. The reflexes were diminished or absent. Fibrillary tremors were observed at an earlier stage in the disease. During the period of observation difficulty in swallowing, with secondary paralysis of the left vocal cord, developed. As a therapeutic measure, 1,500 roentgen units was given over the right brachial plexus, with the left side as a control. Clinically, there was no improvement.

The diagnosis was confirmed by histologic examination of an affected nerve removed for biopsy. References are given to previously reported cases.

R. IRVINE.

Suboccipital Puncture in Cranial Trauma from an Ophthalmic Point of View. Aroldo Pavia, Arch. de oftal. de Buenos Aires **13:** 92 (Feb.) 1938.

Four patients were seen at different intervals after cranial trauma with symptoms of cranial hypertension consisting of intense headache, vertigo, asthenia and disturbances of accommodation. There were no visual disturbances or changes in the fundus. No benefit was obtained from lumbar puncture, but marked relief followed suboccipital puncture, which presents no difficulty or danger. Pavia does not consider that there was any blocking of the medullary canal in these cases. He refers to a report of Magitot in 1936 of 7 similar cases.

C. E. Finlay.

Ocular Muscles

Miner's Nystagmus in the Saar Region. O. Wiedersheim, Arch. f. Ophth. **138:** 515 (May) 1938.

The author, who is director of the ophthalmic clinic for miners in Saarbrücken, has been especially interested in miner's nystagmus for the last ten years. He now gives a comprehensive review of the subject, covering the clinical and scientific, and also the social and economic, aspects of this disease, which affects from 5 to 10 per cent of the coal miners in the Saar. One of the author's original contributions to the study of miner's nystagmus is a photographic method to record the nystagmic oscillations. From the apex of a blackened contact lens a thin metal rod, on the end of which is a shiny button, protrudes in a radial direction. The contact lens is inserted over the cornea of the patient affected with nystagmus, and the excursions of the shiny button are photographed. The most common form of nystagmic excursions in miner's nystagmus is a horizontal or oblique ellipse. The more common frequencies are from 240 to 320 oscillations per minute. The mildest degree of the nystagmus occurs only in the mine (in the dark) if the miner is moving about; the severest degrees are present in daylight even if he is resting in bed. Of great importance is the portion of the field of fixation in which the miner's nystagmus occurs (*Zitterfeld,* field of nystagmus). The onset of the condition is slow and insidious. It usually takes from eleven to fifteen years of work in the mines before the miners become aware of visual difficulties caused by the nystagmus. With regard to the cause of miner's nystagmus, the author agrees with Bartels, Ohm, Randnitz and others in that it is chiefly the insufficient illumination which leads to a disturbance of the delicate neural apparatus that controls the fixation mechanism. Besides, the author believes that the susceptibility to a disturbance of the fixation mechanism varies among different persons and that at the first ocular examination of the prospective miner special attention should be paid to preexisting weakness of the fixation apparatus. The problem of miner's nystagmus is chiefly one of lighting, which can be expected to be solved within the next two decades. Until then, miners with cases of a severe nature should not be pensioned, but their condition should be detected early and they should be given work outside the mine (in present Germany, chiefly roadbuilding and highway construction).

P. C. Kronfeld.

Operations

THE USE OF MUCOUS MEMBRANE IN OPHTHALMIC SURGERY. E. B. SPAETH, Am. J. Ophth. **20:** 897 (Sept.) 1938.

This well illustrated article does not lend itself to abstracting as it deals mostly with technics. Spaeth draws the following conclusions:

"In general, while mucous-membrane grafts have a limited application, these indications are very definite, and, if they are considered carefully, the results obtained should be satisfactory.

"The technique for operating is not difficult; every operating ophthalmologist is capable of the surgery; and most important of all there is no other method available (nor is another necessary) for correcting the defects and the deformities for which the surgery is applicable."

W. S. REESE.

PLASTIC REPAIR OF LID HERNIA WITH FASCIA LATA. B. R. SAKLER, Am. J. Ophth. **20:** 936 (Sept.) 1938.

Sakler describes an operation for the plastic repair of boggy lower lids due to a hernia of intraorbital fat. A fascia lata transplant forms a new orbital septum, anatomically placed and physiologically correct, giving good cosmetic result.

W. S. REESE.

Orbit, Eyeball and Accessory Sinuses

DACRYOETHMOIDITIS: HISTOLOGIC INVESTIGATION AND PATHOGENIC CONSIDERATION. A. POSARELLI, Riv. oto-neuro-oftal. **13:** 555 (Nov.-Dec.) 1936.

Posarelli presents a histopathologic study of the pathways of extension of nasal and paranasal inflammatory conditions to the region of the lacrimal sac. After reviewing anatomic studies of both regions, he describes a number of clinical observations and shows, with the help of photomicrographs, that the extension of the process may take place through venous pathways as well as by continuity and contiguity.

Posarelli goes on to show, as a number of others have shown, that dacryocystitis and peridacryocystitis may have their origin in inflammatory conditions of the paranasal sinuses.

In closing, he proposes on the basis of his studies the name dacryosinusitis in place of the simpler term dacryoethmoiditis.

F. P. GUIDA.

INCOMPLETE SYNDROME OF THE ORBITAL APEX ON A TRAUMATIC BASIS. L. VENCO, Riv. oto-neuro-oftal. **14:** 20 (Jan.-Feb.) 1937.

Venco describes a case of trauma to the left side of the head in which ptosis of the left upper lid, divergence of the left eye and a rapid loss of vision followed almost immediately. Examination showed paralysis of the third nerve but active fourth, fifth, sixth and seventh nerves and the presence of a secondary descending atrophy of the optic nerve. Other neurologic signs were absent.

Roentgenographic examination revealed fracture of the lesser wing of the sphenoid bone at its base, probably by contrecoup, so that it encroached on the optic canal and part of the superior orbital fissure. The mechanism involved was probably one of contusion, compression and laceration of the third nerve and the optic nerve.

F. P. GUIDA.

INFAMMATORY PSEUDOTUMORS OF THE ORBIT WITH UNILATERAL EXOPHTHALMOS: REPORT OF TWO CASES. H. SAUTTER, Klin. Monatsbl. f. Augenh. 100:29 (Jan.) 1938.

After referring to the division of orbital pseudotumors into 3 types by Birch-Hirschfeld, Sautter reports 2 cases of the second type. Operation on patients with this type of tumor shows an absence of a tumor, but the presence of diffused cell infiltration of the orbital tissues and profuse endarteritis. Sautter reports 2 cases, that of a man aged 54 and that of a woman aged 22. Both presented chronic phlegmonous infiltration of the orbital tissues. No genetic cause for the condition could be found; cloudiness of the collateral frontal and ethmoid sinuses in the man was considered too slight to be the etiologic factor, and no pathologic changes were found in the woman. The condition coincided, as to etiology, with that in the other cases cited. Sautter favors the terminology of "inflammatory pseudotumors of the orbit," however, in as limited a manner as possible and not unless histologic and animal research have been done. The author mentions that the condition in his cases differs from that in the 4 cases recently reported by Franceschetti and Rutishauser.

K. L. STOLL.

Physiology

EXPERIMENTAL DATA ON THE PROBLEM OF THE PERMEABILITY OF THE CORNEA. I. M. KLEIN, Brit. J. Ophth. 22:401 (July) 1938. II. M. KLEIN and J. J. SARKANY, ibid. 22:409 (July) 1938.

The first part of this article deals with experiments on excised pigs' eyes and living rabbits' eyes with iodide and nitrite.

The second part has to do with the permeability of the excised cornea with respect to water and chloride ions.

The authors give the following summary:

"1. Under the influence of colloidal osmotic forces a flow of water through the cornea is possible.

"2. This permeability exists not only in the epithelial-endo-thelial direction, but also in the reverse direction.

"3. Chloride ions under the influence of osmotic pressure can diffuse to the opposite surface in both directions."

W. ZENTMAYER.

INTRAOCULAR TENSION AND RETINAL BLOOD PRESSURE AFTER INTRAVENOUS INJECTIONS OF EPINEPHRINE HYDROCHLORIDE. G. SALA, Ann. di ottal. e clin. ocul. 66:9 (Jan.) 1938.

The literature on the effects of epinephrine is reviewed. Nearly all previous observations were concerned with the use of the drug by instil-

lations or by subconjunctival injections. The results were in many respects discordant. The author records observations on 14 patients after intravenous injections of from 0.01 to 0.1 mg. of epinephrine hydrochloride. Results were uniform in the series. No effects on ocular tensions were observed with less than 0.03 mg. With 0.03 mg. a slight rise of tension was observed during the epinephrine crisis, usually 3 mm. (Schiötz), while the increase in blood pressure was marked (from 136 to 186 systolic, for example). With 0.1 mg. of epinephrine hydrochloride, the increase in tension amounted to 7 or 8 mm., while the systolic blood pressure returned rapidly to the previous figures. In 12 cases the retinal arterial pressure was also recorded. This showed a marked rise during the epinephrine crisis, from a normal of 75 systolic and 35 diastolic, for example, to 105 systolic and 60 diastolic after the injection of 0.03 mg. After the injection of 0.1 mg. the pressure rose to 140 systolic and 68 diastolic, dropping rapidly to normal. No visible change in caliber of the arteries was seen, but the veins appeared more engorged, and the appearance of a venous pulse, not previously present, was frequently observed.

S. R. GIFFORD.

Retina and Optic Nerve

NEUROMYELITIS OPTICA. S. H. MCKEE and F. L. MCNAUGHTON, Am. J. Ophth. **21:** 130 (Feb.) 1938.

McKee and McNaughton present the following summary:

"Two cases of myelitis with striking loss of vision are reported. In both there has been ultimate recovery of vision but incomplete recovery from the myelitis.

"In case 1, an adult male, a Canadian, 34 years old, there was a definite history of infection. Visual disturbance, simultaneous in both eyes, and eye discomfort were the first symptoms complained of. These were followed later by transverse myelitis of moderate degree. There were no fundus changes, and recovery was practically complete. The spinal fluid exhibited some evidence of an inflammatory process.

"In case 2, an adult Italian male, 41 years of age, there was no history of infection. Visual disturbance occurred first in one eye and then in the other, with a considerable interval between the attacks. Eye discomfort similar to that present in case 1 was also complained of. The eye symptoms in this case followed the symptoms of myelitis, which were much more severe than in case 1, and resulted in incomplete recovery. Definite fundus changes occurred during the course of the disease. Examination of the spinal fluid showed no change.

"Both cases have been recently examined (April, 1937). The vision, fundi, and fields in each were quite normal. It is of neurologic interest that, in addition to the signs of optic-tract and spinal-cord disease, both cases showed slight signs indicating invasion of the brain stem. In case 1 there was limitation of upward gaze early in the illness; later, lateral nystagmus developed. In case 2, a diminished left corneal reflex was manifested. Such signs have been reported in some cases of neuromyelitis optica."

W. S. REESE.

THE LAURENCE-BIEDL SYNDROME OCCURRING IN A BROTHER AND SISTER. T. K. RATHMELL and M. A. BURNS, Arch. Neurol. & Psychiat. 39: 5 (May) 1938.

The authors feel that although the Laurence-Biedl syndrome is generally known, the disputed etiology makes a report of true syndromes of this type or variants worth while. Several hypotheses as to etiology are mentioned.

The brother and sister described had Russian Jewish parents who were first cousins. The syndrome is based on the factors of familial history, mental deficiency, obesity, retinitis pigmentosa, hypoplasia or inadequacy of the genitalia with distribution of hair characteristics of the opposite sex, phalangeal synostosis, phalanges of the Telford-Smith type and budding of the phalanges.

In Dr. H. E. Riggs' discussion of this paper, she reported the examination of the brain in 2 cases of this syndrome. She states that she has seen evidence in the cerebral cortex and brain stem of widespread arrest in development in early intrauterine life, and her studies suggest that malformation of the visual cortex may play as large a role in the defective vision as does the retinitis pigmentosa.

Burns concludes the discussion, emphasizing the possibility that these patients, 1 of whom reached the eighth grade in school, and the other, the fourth, might have failed to progress farther because of lack of vision. He suggests that if patients of this type could be educated much as are the blind by means of the Braille system, many now considered mentally defective might be educated to take care of themselves.

R. IRVINE.

A CASE OF ANGIOMA RETINAE. I. FEIG, Brit. J. Ophth. 22: 295 (May) 1938.

The unusual appearance of the fundus of the right eye of a woman aged 45 is described. In the region surrounding the papilla there was an outspread network of blood vessels, in which upper and lower layers could be distinguished, partially entangled with each other. In the upper layer, thick blood vessels issued outward in all directions (the thickest of these, which was almost of the thickness of the papilla, pointed downward) and diminished spindlewise in thickness after branching out, so that the network became thinner as it approached the circumference. Below this an entanglement of blood vessels was visible, entwined among those in the upper layer and appearing as rings in certain places. Within a range of approximately six or seven times the width of the papilla nothing but blood vessels could be seen. These were accompanied throughout their length by white reflexes. The periphery showed white areas of degeneration in the choroid. Toward the upper part of the circumference of the fundus there was a gray-green sheen, i. e., a partial surface detachment of the retina.

Feig believes the case to be one of apparently primary angioma which has not led to any other disturbances. Later hemorrhages into the vitreous occurred.

An illustration accompanies the article.

W. ZENTMAYER.

RETINAL HEMORRHAGES BROUGHT ON BY OVERSTRAIN DURING FLYING. J. SEDAN, Ann. d'ocul. **175:** 307 (April) 1938.

The question of physiologic and pathologic vascular reactions to altitude and its variations has become, on account of the rapid progress in aviation, the object of much study and research.

The author, having seen by chance 2 cases of retinal hemorrhages brought on by overstrain at a comparatively high altitude, sought for competent advice on the subject and found it in articles by Beyne, Binet, Bergeret and Flamme. Observations on these 2 cases are given in detail. There is an extensive bibliography.

S. H. MCKEE.

HEREDITARY ATROPHY OF THE MACULOPAPILLARY BUNDLE. R. E. GIQUEAUX, Arch. de oftal. de Buenos Aires **13:** 111 (March) 1938.

Hereditary atrophy of the maculopapillary bundle in 8 members of a single family in 3 generations is reported. In all the members examined the defect was noticed at an early age and was coincident with slight myopia. The ophthalmoscopic picture was that of atrophy of the optic nerve limited to the temporal quadrant. The limits of the visual fields were normal, with a corresponding central scotoma. No general disease was found related to this condition. In some the disease was transmitted by the paternal, and in others by the maternal, line. The disease seems similar to Leber's hereditary atrophy in regard to involvement of both eyes and in its nonprogression after reaching its maximum; it differs in a more precise central localization, in localization of the atrophy to the temporal quadrant and in its earlier appearance.

C. E. FINLAY.

PRESENT STATUS OF THE OPERATIVE TREATMENT OF RETINAL DETACHMENT. C. E. FINLAY, Rev. cubana de oto-neuro-oftal. **6:** 24 (Jan.-Feb.) 1937.

The present status of the operative treatment of retinal detachment is reviewed. The author points out that a modification of Gonin's original operation devised by him several years ago is practically the same as the modern operation of diathermy. In this operation subretinal puncture was subsequently performed and the order of the steps was reversed; also galvanocautery was employed in place of diathermy. In this paper the author stresses Pavía's method of localization of the tear by external illumination with a small lamp fixed at the end of a long probe, and the observation of its effect ophthalmoscopically.

C. E. FINLAY.

Endoangiitis Obliterans: Report of a Case. H. Geserick, Arch. f. Ophth. 138: 647 (May) 1938.

In a German blacksmith who had taken part in the World War there developed in 1921, when he was 28, visual disturbances which were found to be caused by recurrent retinal hemorrhages. In 1925 he was admitted to the ophthalmic clinic of the University of Jena, and the diagnosis of choroiditis of tuberculous origin and of retinitis proliferans of both eyes was made. Under treatment with tuberculin the fundi showed signs of temporary improvement, but later the vascular disease became progressive again and atrophy of the optic nerve set in. In 1934 the fundi presented the picture of extensive obliterating endoangiitis with mild choroiditic changes. In addition, a serious nervous disease developed which was characterized by multiple lesions resembling those seen in multiple sclerosis. The pulsation in the dorsalis pedis arteries could not be felt. The author interprets this case as one of thromboangiitis obliterans. This condition was first considered as cause of retinal periphlebitis by Marchesani (*Arch. f. Augenh.* 109: 124, 1935). On the other hand, Oppenheim in his "Lehrbuch der Nervenkrankheiten" (Berlin, S. Karger, 1913) first mentioned cases of multiple sclerosis caused by "a general vascular disease." The conclusion is justifiable that the fundus picture of retinal periphlebitis or of retinitis proliferans does not always indicate a tuberculous disease of the wall of the vessel. The cause of Buerger's disease is still unknown.

P. C. Kronfeld.

Trachoma

Frequency of Parenchymatous Vessels in the Cornea of Patients with Trachoma. A. Busacca, Arq. brasil. de oftal. 1: 5 (June) 1938.

After reference to the literature on the situation of the newly formed blood vessels in trachomatous pannus with respect to Bowman's membrane, the author reports the result of the biomicroscopic examination of 100 eyes of 50 patients with trachoma. He finds blood vessels in the corneal stroma in 44 per cent of the patients and in 34 per cent of the eyes examined.

He classifies these vessels according to their origin into vessels proceeding from the limbus and into vessels originating in the pannus and according to their manner of distribution into simple or scarcely ramified and reticular vessels.

He discusses the differential diagnosis of syphilitic keratitis. Busacca finds these parenchymatous blood vessels even when the pannus is not much developed. In the cases in which they are found, the trachoma is more severe and less amenable to treatment.

C. E. Finlay.

The Latest Result of Etiologic Research of Trachoma. L. Poleff, Klin. Monatsbl. f. Augenh. 99: 584 (Nov.) 1937.

Poleff presents a detailed résumé of recent research on trachoma, done by Szily, Busacca, Cuénod and others. The rickettsias, resembling the inclusion bodies of Prowazek and Halberstädter, have been described

almost identically by Busacca and Cuénod. Cuénod and Nataf found a "genuine culture of rickettsias" in the digestive tract of lice which they inoculated. These authors proved definitely the identity of the germ of trachoma and of Rickettsia rocha-limae, a parasite of the vestment house. A watery solution containing dirt from the nails of a small girl, infested with lice, was instilled into the eyes of a Maccacus rhesus; trachoma developed, and rickettsias were found in the lacrimal fluid. Poleff succeeded in growing tissue of trachoma in vitro. He obtained tissue cultures of conjunctival trachoma follicles and corneal pannus. These infrabacterial bodies, resembling those described by Cuénod and Nataf, could be cultured by Poleff under certain conditions. He could prove, furthermore, that cultures of rickettsias affected the eyes of rabbits pathologically; they produced intraocular formation of follicles, followed by atrophy of the eyeball, when they were introduced into the vitreous and descemetitis when they were introduced into the anterior chamber. Poleff gained the conviction from his experiments with pure cultures of rickettsias that they are identical with the formations described by Cuénod and Nataf and that they are the cause of trachoma.

K. L. Stoll.

Tumors

Cylindroma of the Orbit. Carlos S. Damel and E. R. Villegas, Arch. de oftal. de Buenos Aires **13:** 62 (Feb.) 1938.

A case of orbital cylindroma arising from the lacrimal gland is reported. Krönlein's operation was performed, and histologic examination showed the growth to be a cylindroma of the lacrimal gland.

C. E. Finlay.

Protrusion of the Eyeball in Carcinoma of the Parotid Gland with Metastases at the Base of the Skull: Report of a Case. H. Heymann, Klin. Monatsbl. f. Augenh. **100:** 23 (Jan.) 1938.

Heymann reviews the various types of orbital carcinoma and the literature in point. His own case was that of a woman aged 39, who had keratitis due to lagophthalmos of the right eye in February 1937. The left eye was free from congestion, and vision was normal. The patient had suffered from chronic otitis media since 1931, which resulted in paresis of the facial nerve in 1933. The right eye protruded; the measurements with Hertel's apparatus were 20 mm. in the right eye and 18 mm. in the left. The parotid gland, swollen since 1931, was treated with roentgen rays in 1934. The protrusion increased after the patient's admission to the hospital, but the disk remained normal. The result of detailed examinations suggested that the tumor grew from the parotid gland upward through the bone into the middle cranial fossa, then anteriorly and medially toward the right orbit. This tumor was a solid carcinoma containing basilar cells.

K. L. Stoll.

Uvea

UVEOPAROTITIS WITH PERIVASCULITIS AND OTHER RARE SYMPTOMS: REPORT OF A CASE. GUNNAR VON BAHR, Acta ophth. **16:** 101, 1938.

Swelling of the lacrimal and parotid glands, iridocyclitis, a few small, gelatinous iris nodules and fever developed in a 42 year old man. There were also present in the upper eyelids several small, compact tumors. On excision, these proved to be composed of typical tubercle-like granulation tissue at the site of tubules of the accessory lacrimal glands (Krause's glands). Inoculation of this tissue into a guinea pig gave no results. The most interesting and unusual feature of the case was the presence of a mild perivasculitis, as manifested by a narrow, grayish white sheathing around some of the retinal veins and in one place, around an artery. This perivasculitis disappeared entirely in a few weeks, as did the iris nodules.

O. P. PERKINS.

Vision

VISUAL ACUITY OF NOMADS IN ASIA MINOR. S. IRMAK, Deutsche med. Wchnschr. **64:** 677 (May 6) 1938.

A discussion of the various methods of determining visual acuity and the means of deciding what is the angle subtended introduces the article. The author concludes from his work with the nomads in Asia Minor that they have the keenest vision of all peoples. In one patient vision was nine times the normal. The visual angle for the minimum visible subtends 5.5 seconds; the retinal image, 0.34 millimicrons. Even in the aged a vision of twice or three times normal is frequently found. Good vision seems to be a dominant characteristic. A bibliography accompanies the article.

L. L. MAYER.

CENTRAL SCOTOMA IN CONGENITAL AND STRABISMIC AMBLYOPIA. E. HAITZ, Klin. Monatsbl. f. Augenh. **99:** 761 (Dec.) 1937.

Heine proved that unilateral amblyopia without pathologic changes was usually caused by a central scotoma, which he found in 90 per cent of the patients he examined. Uhthoff found a central scotoma in only 50 per cent of 100 patients, because he eliminated those whose vision was less than one sixth of the normal. Haitz has devised a binocular stereoscopic method with which he and a number of other ophthalmologists were able to show far smaller scotomas than they could demonstrate with other methods. The diameter of the scotomas varied between 2 and 6 degrees; their shape was usually round, and they were often connected with the blindspot. The paracentral scotomas were usually located temporally from, and close by, the center. The peripheral outline of these fields was usually normal for white and colors.

K. L. STOLL.

Miscellany

AN ACCOUNT OF THE OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM *

L. VERNON CARGILL, F.R.C.S., LONDON, ENGLAND

REVIEWED BY BURTON CHANCE, M.D., PHILADELPHIA

The *Medical Press and Circular* for some time past has published several series of articles sketching the history of British medical societies. In the January 1938 issue there is an account of the Ophthalmological Society of the United Kingdom, prepared by L. Vernon Cargill. So important has this society been in the modern development of ophthalmology that the ARCHIVES deems it fitting that younger readers should know somewhat in detail the chief events of its history. It is well that the causes of the needs for such an organization should be viewed in retrospect.

In the earliest years of the nineteenth century the practitioners in England who treated diseases of the eye treated general diseases as well, yet it was likely that they were especially skilful only in certain maneuvers for the relief of particular ocular conditions. In consequence, long before the middle of the century a number of surgeons had attained celebrity, although the knowledge they had acquired was based on what they had found by their study solely of the external features of the ocular globe and the orbit. Cataract and glaucoma could be understood at most only as viewed by the naked eye, with the help, perhaps, of the condensing lens, as instituted by Mackenzie, for not until 1851 was the ocular fundus visible during life.

The invention of the ophthalmoscope brought about a marvelous revelation, yet it was not for several years that many observers in England were competent to explain what to them had become visible. By the average user of the instrument, ophthalmoscopy seemed difficult of comprehension, yet because of the seeming complexity and because of what they expected from the shadows of what they feared might ultimately lead to blindness and destruction of the globe, men determined to master the intricacies of the investigation. In the succeeding twenty years, through the intensification of their studies which had been induced by the use of the ophthalmoscope, knowledge of the facts relating to ocular diseases became more widely disseminated and their seriousness appreciated. Many who had been practicing general surgery gradually became more engrossed in the comprehension of ophthalmic problems and steadily allowed other subjects to recede from the horizon of their daily practice.

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* Cargill, L. V.: Ophthalmological Society of the United Kingdom, M. Press **196**:4 (Jan. 5) 1938.

The fact of this specialization aroused in the minds of the foremost physicians in Great Britain a desire to form some sort of union for mutual recognition and fellowship and for the furtherance of scientific investigation and more exact practice.

The Royal London Ophthalmic Hospital, established by Saunders so early as 1804, and "dispensaries for curing diseases of the eye and ear" were in existence in various parts of the United Kingdom, yet for many years Moorfields and other special hospitals were regarded somewhat askance, in spite of the honorable work which had been accomplished by Farre, Saunders, Travers, Lawrence, Tyrell, Dalrymple, Guthrie, Mackenzie, Middlemore and others. The word ophthalmology had hardly become current, and so late as 1868 ophthalmic practice had no special service assigned it in the schools or by the general hospitals.

The activities at the Royal Ophthalmic Hospital, soon to be called and still affectionately remembered as Moorfields, had from the start attracted and held many who were determined to create a true division of medicine, so swift and brilliant was the progress that followed the introduction of the ophthalmoscope. The revolution which the use of that instrument instigated brought the range of ophthalmology within the scope of general medicine, and workers in England were keeping pace with the strides set by investigators on the Continent; the subject of ophthalmology henceforth became of vital interest in medical circles.

Moorfields at that time was located in the region of Finsbury, then a wealthy residential section, and many young medical men resided there. It became the custom for those who were interested in ophthalmology and who resided around Moorfields on certain evenings in the week to gather at the hospital, in the house surgeon's room, to discuss cases and other matters in which they were interested. A sort of Moorfields Club was formed; so far back as 1866 Jonathan Hutchinson records in the *Ophthalmic Hospital Reports* how he read a paper at such a meeting.

At one of those informal meetings early in 1880 it was suggested that an ophthalmologic society be formed. A circular was drawn up and sent to the leading ophthalmic surgeons in the three divisions of the United Kingdom, inviting them to help form a society. It met with a cordial response, and a preliminary meeting was held in June of that year, which was attended by 28 of the leading men among ophthalmologic practitioners. A society was then formally organized, and William Bowman was acclaimed president, as it was chiefly through his efforts that the society was founded. Bowman, because of his earlier investigations into the anatomy and physiology of the kidney, was deemed, in the parlance of the time, to be the "Father of the Kidney"; he might also be considered as the "Father of British Ophthalmology."

At another meeting a set of rules was adopted which were subscribed to by a list of 63 members. An inaugural meeting was held in October. The membership doubled by further admissions. The youthful organization comprised such well known men as George Critchett, Hughlings Jackson, Jonathan Hutchinson, Argyll Robertson, Brailey, Brudenell Carter, Thomas Buzzard, Couper, Nettleship, Gowers and Waren Tay. At the end of the first year the ordinary members numbered 141, and Donders and Helmholtz were elected to honorary membership.

During 1880 eight meetings were held in the rooms of the Medical Society of London on Chandos Street. Color blindness, the relation

between optic neuritis and intracranial disease, the ocular symptoms in locomotor ataxia and retinal hemorrhages associated with high arterial tension and also occurring in patients suffering from pernicious anemia were among the topics discussed. Tay reported a case under the title, "Symmetrical Changes in the Region of the Yellow Spot in Each Eye of an Infant." McHardy exhibited "electromagnets for the detection and removal of fragments of iron and steel from within the eye."

The society immediately became the rallying ground of the best practitioners and students in the United Kingdom and soon began to attract the attention of the medical world. At the end of the second year 158 members were enrolled, and the communications numbered 71.

Jonathan Hutchinson succeeded Bowman as president and maintained his membership for years, constantly contributing to the *Transactions.* He presided over the society for three years. In his valedictory address he called attention to the progress which was then being made toward accuracy in clinical and pathologic knowledge, and he lauded the introduction of cocaine, which was used as a local anesthetic at Moorfields for the first time in September 1884.

In 1883 two events of great importance to the society occurred. The Medical Society had made extensive improvements in the premises and, in consequence, the rent for the rooms used by the Ophthalmological Society was increased. The finances of the society were found to be inadequate for that added expense. Bowman generously undertook to bear the cost of all fittings necessary for a museum and library, besides making a gift of £50 annually for twenty years! These benefactions placed the society in a sound financial position. A library was founded in 1884 with about 100 books and continued to increase both in size and in importance. In 1887 a catalog was published under the direction of Adams Frost, who should be remembered for his "Atlas of the Fundus Oculi"; Frost continued as librarian for several years.

The society sought from the beginning to influence the medical authorities to regard ophthalmology as a legitimate specialty, yet it was not until 1891 that courses in the fundamentals of ophthalmic medicine and surgery were established in the medical schools, so from that date all students have been required to attend hospital clinics and courses of systematic lectures on ophthalmology.

It was significant in this respect that Mr. Argyll Robertson, who became president in 1893, referred to the great service which ophthalmology had already rendered to general medicine and surgical therapeutics and pleaded for a firm union of ophthalmic specialists with each other and with general practitioners. Nettleship urged that specialists should be attached to special hospitals.

At the suggestion of Dr. William Gowers, an active member for years, the Bowman Lecture was founded, quite without the knowledge of Bowman, in commemoration of his valuable service to the Ophthalmological Society and in recognition of his distinguished scientific position in ophthalmology and in other branches of medicine. The lecture was to consist of a résumé of recent advances in ophthalmology or of any original investigation, to be delivered at a special meeting called for that purpose only. The first lecture was delivered by Jonathan Hutchinson on Nov. 13, 1884, his subject being "On the Relation of Certain Diseases of the Eye to Gout."

There have been 28 Bowman Lectures. Among the foreigners who have delivered them have been Zehender, Hansen Grut, Leber, Snellen, Fuchs, Sattler, Landolt, Uhthoff, Morax, de Schweinitz and Van der Hoeve.

The interest of the society was aroused early in the prevention of blindness from ophthalmic neonatorum, and it was urged that the attention of the medical press and those engaged in the practice of obstetrics should be called to this important matter. A legal enactment compelling local government boards to warn against the dangers of the disease was sought. But the society was advised that there was no legal power yet established by which the local boards could compel attention to the danger, although boards of guardians had always shown a readiness to comply with the suggestions presented for the health of the sick.

In the spring of 1892 the society met a severe loss in the death of Sir William Bowman. So vitalizing had been Sir William's influence and patronage over the society that at his passing there seemed to have been created a hiatus the contemplation of which caused the members to pause and consider how they might be able to go on in the future without his companionship. Hansen Grut, of Copenhagen, when introduced as the lecturer in 1890, said:

> Those of an older generation had had one great advantage not enjoyed by his younger colleagues, for they had witnessed the glorious dawn of modern ophthalmology. In the older men's first years of study and practice they were led by such geniuses as von Graefe, Donders, Arlt, von Jaeger, Critchett and, besides many others, Sir William Bowman, who had always stood as the great example to the practitioners of ophthalmology. Foreigners fully understood and shared the unparallelled love and admiration which was bestowed upon Bowman by his countrymen, for he had served for years as the glorious illustration of what the great men of the grand period of Ophthalmology were like.

In 1901 the Edward Nettleship Prize for the encouragement of scientific ophthalmic work was inaugurated by the friends and pupils of Nettleship. The prize consists of a gold medal and has been awarded at intervals of three years. In addition to provision for the medal, surplus funds are used in the purchase of works for the library.

In 1913 the library was housed in a separate room in the quarters of the Royal Society of Medicine, and the additional privilege of holding meetings in its rooms was extended to the Ophthalmological Society.

The dues of the society were originally 1 guinea, but in 1920, owing to the increased cost of the *Transactions,* the dues were increased to 1½ guineas. An Illustration Fund was formed to relieve authors of this expensive item in the publication of their contributions. The *Transactions* have been published annually, and besides the individual indexes, additional complete indexes have been prepared at intervals of ten years.

There had been agitated for several years the proposition that the ophthalmologic section of the Royal Society of Medicine should unite with the Ophthalmological Society, but in 1912 it was decided that no such union should be formed because the society was unwilling to lose its identity and its independence.

In 1917 it was decided that other ophthalmologic societies in the British Empire might become affiliated and that their proceedings might be published in the *Transactions.* The *Transactions* of 1919 contained

reports of five affiliated societies: the Oxford Ophthalmological Congress; the Midland, the North of England and the Irish Ophthalmological Society, and the Ophthalmological Society of Egypt. Later, the Scottish Ophthalmological Club was added to the group. In 1924, when Egypt became a sovereign state, the Egyptian Ophthalmological Society was no longer affiliated. In 1927 the South Western Ophthalmological Society became affiliated.

The meetings have usually been held in London, but Glasgow, Liverpool and Edinburgh have been honored. In the early years of its existence, the meetings of the society were held several times in the year, but in 1913 it was decided to hold an annual meeting of several days' duration. In 1925 the meeting took the form of a convention of English-speaking ophthalmologic societies. At this convention the minister of health and the American ambassador made addresses, and there was presented to the society the Critchett Memorial Presidential Badge, to be worn by the president of the society, offered by the family in memory of George Critchett and Anderson Critchett, father and son. Mr. Treacher Collins, the incumbent of that year, who had been largely instrumental in the success of the convention, was the first to wear the badge. In addition to the interesting and comprehensive program, the members visited the institutions in London, and at night a dinner was held at the Guild Hall. At a general meeting a committee was appointed to obtain cooperation for the promotion of international congresses, and this led to the resumption of the congresses which had been discontinued during the World War.

In 1932 the society lost one of its most distinguished members, Mr. E. Treacher Collins; he had been president twice, had been a Bowman lecturer and had received the Nettleship Medal. He bequeathed £500, which has been added to by subscriptions by others for funds for the Treacher Collins prize essay.

A fifty years jubilee was celebrated at the annual congress of the society in London in 1930, at which many distinguished foreign ophthalmologists were guests. The Bowman Lecture was delivered by Sir Arthur Keith, who proclaimed Bowman as one of the greatest anatomists England has produced.

That the society, which is active and virile, will take as important a part in "the cultivation and promotion of ophthalmology" in the future as it did in the past is assured by the membership composing it. According to Dr. Cargill, in 1937 there were besides life and honorary members, 571 general members. Only one original member, Sir Lindo Ferguson, of New Zealand, was still living.

Society Transactions

Edited by W. L. Benedict

FRENCH OPHTHALMOLOGICAL SOCIETY

Fifty-First Congress, Paris, May 16-19, 1938

First Session, May 16

Double Trephining with the Aid of a Trephine with a Movable Crown in Dacryorhinostomy (Lantern Slides). J. Subileau, Paris.

The success of dacryorhinostomy depends to a large extent on the correct trephining of the lacrimal duct. The trephining should be wide. The author uses a trephine inspired by the two trephines of Arruga, which he combines into a single instrument. A crown with teeth slides around a cylinder furnished with a catch, which serves as a pivot. The crown is automatically locked by means of a bayonet device. In the first position the central pivot slides forward and passes the teeth. It rests on the bone to permit the making of a tracing. In its second position the pivot disappears, and trephining is accomplished by driving the crown into the tracing line. The diameter of the trephine is 8 mm.

The author prefers a double trephining, one opening being overlapped on the other more or less according to the size of the lacrimal sac, to the single trephining, with which one runs the risk of leaving the bony promontories obstructing the operative incision by hindering the eversion of the lips of mucous membrane. With the double trephining the opening is the shape of an 8. It is completed in front by the aid of a Citelli forceps. The method permits a more rapid operation and has the advantage of sparing the patient the shock due to the use of the chisel and mallet.

Roentgen Therapy for Meibomian Epithelioma. Pierre Dupuy-Dutemps, Paris.

Roentgen therapy was used successfully in 2 cases of meibomian epithelioma. The cure was of long enough duration to be considered permanent.

Though wide excision at the onset remains the best means of treating cutaneous epithelioma of the ocular region, it seems that the application of the roentgen rays is the method of choice for glandular epithelioma of the eyelids.

Symmetric Lymphoma of the Two Semilunar Folds: A Symptom Indicative of Lymphatic Leukemia (Lantern Slides). Marcel Kalt and H. Tillé, Paris.

A man aged 66, in good health, who sought consultation for watering of the eyes, was found to have a purplish blue tumor the size of a bean at the level of each semilunar fold; the growths did not involve

the caruncle and extended under the epithelium of the conjunctiva of the inner cul-de-sac. Biopsy of the growth on the right revealed it to be a typical encapsulated lymphoma. Three other lymphomas existed under the buccal mucous membrane. There was moderate polyadenopathy, and the spleen was hypertrophied. Chemical studies of the blood revealed lymphatic leukemia with leukocytosis, the white cell count being 65,000 (92 per cent large and medium nongranular mononuclear cells and 3 per cent lymphocytes).

Since the patient's general condition was good, treatment with an arsenic preparation was given for a month. Three weeks after its discontinuance, there appeared another lymphoma of the right bulbar conjunctiva, bilateral hemorrhages of the retina (without leukemic retinitis), epistaxis, purpura, asthenia, an increase of the adenopathy and the splenomegaly and an increase in the globular anemia and the leukocytosis. In view of this disturbance in equilibrium, roentgen therapy of the spleen and of the ganglions was indicated—a two-edged weapon that had been kept in reserve on the advice of Dr. Pagniez.

The development of the leukemia in this case and the fate of the semilunar lymphoma on the left side (left as a control) will be published subsequently.

Experimental Study of the Cicatrizing Action of Insulin on Corneal Wounds. C. Dejean and P. Artières, Montpellier.

This study was made on 34 rabbits of an average weight of 3,000 Gm. It consisted in comparing the rapidity of corneal cicatrization in groups of rabbits, some of which were subjected to therapy with insulin while the others served as controls. The same type of corneal wound was made in all the animals by means of Elliot's trephine. The conclusions of the authors are as follows:

1. In the case of a rabbit weighing 3,000 Gm. with a loss of superficial corneal substance a daily injection of from 10 to 15 units of insulin advances by two to four days the cicatrization of a superficial corneal defect with regular edges.

The addition of sugar to insulin is necessary to avoid hypoglycemia.

2. Under the same conditions a dose of 5 units is insufficient to activate cicatrization.

3. Local applications of insulin in an ointment do not seem to accelerate cicatrization under the conditions mentioned.

Hypopyon Keratitis and Hypertonia. A. Motolese, Florence, Italy.

The author reports experiments which confirm the role of hypertonia in the development of hypopyon keratitis.

In their entirety, the results of these experiments prove that hypertonia plays one of the most important roles in the development of hypopyon keratitis.

In fact, the author was able to show that the progress of the corneal ulcer was accelerated by the hypertonia which was induced experimentally; that in all the cases considered more or less complete destruction of the cornea was observed, whether virulent germs previously taken from pure cultures or less virulent germs previously taken from recent cultures were employed; and finally that the ulcer could be made worse by the induction of hypertonia during its development.

The treatment which Bardelli ordinarily applies to this disease in the ophthalmologic clinic of Florence is reported.

INFLUENCE OF RADIUM ON INTERSTITIAL KERATITIS (Lantern Slides). W. J. KAPUSCINSKI, Posen, Poland.

The action of radium on the course of interstitial keratitis manifests itself by the following signs: (1) rapid vascularization and just as rapid disappearance of newly formed vessels, (2) reappearance of opacities after their initial disappearance, (3) dispersion and migration of the opacities and (4) clearing up of the cornea, at times rapid and permanent.

The best dose is 66 mg. of radium element with a filter of 1 mm. of platinum at a distance of 2 cm. for six hours. This dose does not injure the cornea.

SURGICAL TREATMENT OF DISEASES OF THE CORNEA. R. RUBBRECHT, Brussels, Belgium.

The method recommended is applicable to most of the superficial diseases of the cornea, that is, those which concern only the epithelium and the anterior lamellae of the parenchyma. The operation consists in excising the diseased corneal focus and in covering the loss of the substance thus caused by means of a thin section taken from the bulbar conjunctiva. This section should be limited as much as possible to the epithelial bed. The esthetic and visual results will be all the better in that the subepithelial dermal bed is thinner. By this operation one can rapidly and permanently heal stubborn corneal lesions. The transplanted section constitutes a solid protection, preventing recurrence. It is compatible with a high degree of transparency.

DEVELOPMENT OF KERATOCONUS (Lantern Slides). MARC AMSLER, Lausanne, Switzerland.

Keratoconus is regarded as an evolutive and progressive condition. Of a total of 42 patients (from which group are excluded those with keratoconus classified as of the fourth degree) followed during a period of from two to nine years, only 6 showed an increase in the characteristic deformation on keratography. There was a gradual progression of keratoconus, occurring at different ages (about 25, 30, 40, 50, 53 and 60). In the great majority of instances the latent or mild keratoconus has been found to be stationary, at least within the limits of the time of observation.

Keratoconus of the fourth degree often gives rise to more or less inflammatory and acute changes, which, however, are incidental, transitory and curable.

FIBROMA OF THE SCLERA (Lantern Slides). M. TEULIÈRES and J. BEAUVIEUX, Bordeaux.

The authors report the case of a 5 year old child afflicted with a fibroma of the sclera, which had probably originated in the tendon

of the superior rectus muscle, the rapid evolution of which had suggested that it was a malignant tumor.

At operation a tumor the size of a small chestnut, intimately united to the sclera in its anterior portion, was excised through the upper cul-de-sac of the conjunctiva. The shape and movements of the eyeball and the visual acuity were restored.

The anatomicopathologic examination revealed that the growth was a pure fibroma.

Iritis and Focal Infection: Allergy; Reticuloendothelial System; Clasogénines (Lantern Slides). Henri Lagrange and Jean Goulesque, Paris.

The pathogenic problem raised by the localization and subordination of certain pathologic reactions occurring as manifestations of a latent infectious focus is considered. Reactions such as these produce forms of iridocyclitis which constitute the nosologic group of iridocyclitis due to focal infection. The authors show that the condition is a manifestation of tissue allergy, the localization of which is determined by the presence at the level of the ciliary processes of an important reticuloendothelial cellular colony.

Heerfordt's Syndrome: A Special Form of a New Extensive Reticuloendotheliosis; Besnier-Boeck-Schaumann Disease. L. M. Pautrier, Strasbourg.

Pautrier points out that the syndrome of uveoparotitis, called Heerfordt's syndrome, has been considered until now as an essentially ophthalmologic disease, in spite of the syndrome of infectious parotitis, which forms part of it, and of the different symptoms associated with it, that is, recurrent facial paralysis and cutaneous eruptions, to which until now no one has called attention systematically and above all connected with the same process.

Pautrier has been able to prove in a series of recent publications that in reality the syndrome of Heerfordt should be placed in the category of a new extensive reticuloendotheliosis isolated by dermatologists, called Besnier-Boeck-Schaumann disease, of which it constitutes only a special form. For the support of his argument he recalls a recent case which he reported in the *Bulletins et mémoires de la Sociéte médicale des hôpitaux de Paris* (53: 1608 [Dec. 20] 1937) as well as the cases of Lesné and Coutela, of Lamy and of Paufique. The authors seem, indeed, to agree with reference to the classification.

Besnier-Boeck-Schaumann disease is characterized by cutaneous sarcoid eruptions and pulmonary, ganglionic, osseous, glandular, visceral, ocular and neural lesions. Its manifestations may be distributed in the whole reticuloendothelial system. The lesions, wherever their site, have a constant anatomicopathologic unity and are made up of squamous epithelioid cells, at times with the addition of giant cells but without tuberculous follicles, foci of caseation or the presence of the bacillus of Koch.

The same histologic structure is present in this disease that is present in Heerfordt's syndrome, in parotitis, in uveal lesions and in neural

lesions. The identity of the two clinical and histologic types is beyond doubt.

The tuberculous origin scarcely seems likely because of the spontaneous healing of all these lesions. If vision at times remains impaired in Heerfordt's syndrome, it is because of the complexity and delicacy of the eye, which cannot meet the requirements for complete recovery. It seems more probable that the disease is caused by a virus which is yet to be isolated. Additional researches are necessary to determine the cause.

Post-Traumatic Pearly Cyst of the Iris: Inclusion of an Eyelash in the Anterior Chamber (Lantern Slides). M. Teulières and J. Beauvieux, Bordeaux.

A pearly cyst, similar to those which Masse has been able to produce experimentally, developed on the anterior surface of the iris after the penetration of an eyelash into the anterior chamber at the site of an accidental wound.

The cyst when removed showed the normal structure of tumors of this kind; that is to say, the cavity was lined with stratified pavement epithelium of the malpighian type.

Entoptic Pupilloscopy: Spontaneous Variations of the Pupillary Diameter. Henri Viallefont and R. Lafon, *Montpellier.*

By the entoptic method, Viallefont and Lafon have established the fact that in the absence of exciting factors the mean diameter of the pupil is constantly variable, these variations being absolutely independent of the pulse, of respiration and of psychic modifications. A graphic representation of the pupillary diameter would be expressed as an irregular broken line.

Second Session, May 17

Shock Therapy in Ophthalmology. L. Hambresin, Brussels, Belgium.

The work of Hambresin is divided into four extensive parts. In the first part he deals with the phenomena of shock. Shock is a physiologic imbalance the beginning and termination of which are abrupt. It rests only on functional disorders and is not followed by any lasting anatomic lesion. Occurring under many circumstances, it has a clinical picture which is far from being univocal. However, there exists always a disturbance of the blood, resulting in what is called the hemoclastic crisis, the principal symptoms of which are arterial hypotension and leukopenia.

The second part has reference to shock therapy, which aims at checking the evolution of a disease by provoking an artificial crisis, an artificial shock. One speaks of great shock, of attenuated shock, of "formes frustes" of shock and even of local shock, according to the importance of the phenomena which accompany it.

Shock therapy is of use for two groups of pathologic conditions. The first group consists of all diseases which show at the beginning a state of plasmatic instability, such as hemophilia, paroxysmal hemo-

globinuria and anaphylaxis. In the presence of such conditions shock reestablishes normal equilibrium. It serves as a desensitizing measure. The second group includes the infections. In cases of such conditions the secondary effects of shock are utilized to check the progress of the disease; in such cases shock treatment actually increases the defense of the organism.

In the third part all the agents of therapeutic shock are reviewed. The substances which are currently employed are the organic substances (peptone, milk and blood), metals and metalloids in the colloidal state (sulfur, silver and gold) and microbes. All the vaccines contain microbes. In the field of ophthalmology one makes special use of dmelcos (a vaccine of Ducrey bacilli), of Renard's vaccine, of propidon (a mixed stock vaccine prepared by the method of P. Delbet) and of the vaccine triple V (a bacterial vaccine containing the organisms of typhoid and the two paratyphoids).

After having explained in detail the use of all these products, the author takes up the "fixation abscess," which is considered by many clinicians as a method of shock therapy. The waste products resulting from the disintegration of the tissues become heterogeneous albumins, the reabsorption of which produces humoral modifications. He has also reserved a paragraph for pyretotherapy, which often only determines shock phenomena, and to a study of injections of charcoal suspension.

In ocular therapy use is made of all these agents, except peptones, by the intravenous route. However, most often milk is employed, milk brought by the milkman and not the substitutes placed on the market by manufacturers of pharmaceutic products, which are all less active. The milk should be fresh. It should be boiled for from eight to ten minutes and injected at once after boiling, as soon as it has returned to normal body temperature. The injection is made under the skin at the level of the flank. Five cubic centimeters is used for the first injection. According to the results obtained, one continues to use this quantity or to increase the dose. The amount used should not exceed 10 cc. From three to four injections are made at intervals of two or three days. In view of the sensitivity of certain patients, it is always prudent to make a preliminary injection of 1 cc. an hour before the first puncture.

The principal contraindications to milk therapy are tuberculosis, cardiac diseases and arteriosclerosis. Neither is the method justified in a case of pregnancy. As for accidents, the formation of abscess, manifestations of delirium and the phenomena of anaphylaxis may occur. The literature records also some fatal cases. The study of these cases shows that in infants there were several recurrences of tuberculous lesions and that the death of adults should be ascribed either to shock or to infection. In the cases in which shock seems to have been the cause of death, the patients were always asthmatic.

The last part of Hambresin's work is reserved for the study of ocular diseases treated with shock. Almost all ophthalmologists make use of this method. The successes reported are numerous and include the most varied diseases. One can say, nevertheless, that the best results are obtained in the diseases of the anterior segment of the eye. The

main conditions in which the treatment is indicated are: gonorrheal conjunctivitis, hypopyon ulcer, iritis, cyclitis and infected wounds. Among these indications, the blennorrhagic inflammations of the conjunctiva are the principal ones. Milk is the best shock-producing agent in cases of gonorrheal conjunctivitis in adults. In case of corneal complication, treatment with milk should be instituted at the onset. As for gonorrheal conjunctivitis of newborn infants, in the opinion of many authors therapeutic shock is far less satisfactory.

Galactotherapy often works wonders for the keratoconjunctivitis of infancy. It diminishes the reactive phenomena of parenchymatous keratitis. In the treatment of sympathetic ophthalmia it seems that injections of milk should give way to injections of turpentine.

As to his conclusions, the author insists first of all on the fact that the production of shock is not a specific treatment. Its essential mechanism is unknown. It probably involves an extremely complex action. Shock seems to give a stimulus which puts into play the defense mechanisms of the organism, among which the reticuloendothelial system occupies one of the first places. Although one might say that focal reaction constitutes the main point in the therapy, hyperthermia is useful. The finest results are obtained, without doubt, when there has been a high fever. In a general way, the results of shock therapy are much more favorable in acute localized processes than in acute generalized diseases. The method, however, offers interesting results in chronic diseases, especially when they are confined to an infectious focus. The fact that the lesion is localized constitutes, accordingly, a favorable condition for success. It is thus that the numerous successes in the treatment of ocular diseases are explained.

DISCUSSION

E. Aubineau, Nantes: Dr. Hambresin is right in considering even inactive tuberculosis as a contraindication of shock therapy. I should like to call his attention to the case of a girl of 18 years afflicted with "recurring retinal hemorrhages" and with pleurisy of long standing, who was treated with shock by means of injections of serum. Good visual results were obtained, but pulmonary tuberculosis developed, clearly as a result of the shock, and death ensued.

Long and Extensive Experience with Shock Treatment by Intravenous Injection of Electraurol. A. Jacqueau, Lyon.

Jacqueau has given more than 3,000 intravenous injections of electraurol (a proprietary electrocolloidal gold solution). He has never had an accident or even an untoward occurrence. The dose employed has always varied between 2 and 5 cc. This dose can be repeated on several consecutive days. Noticeable shock is often absent. The injection is painless.

The indications are those for humoral therapy, called shock therapy. Patients operated on for cataracts are the first to benefit by the treatment. One must act promptly at the least suspicion of endogenous infection, even at the least indisposition of the patient. In cases of infectious iritis with hypopyon and in cases of disturbances of the vitreous of the same nature, its action is rapid and effective. The same

holds good for intraocular infection resulting from sclerectomy. The indication is clearcut in cases of ocular perforations with menacing panophthalmia. Sympathetic ophthalmia seems to be much improved by prolonged treatment. Inflammatory lesions of the corneal or the scleral wall and lesions of the eyegrounds are not influenced or scarcely so.

Typhoid Vaccine in Ocular Therapy. W. J. Kapuscinski Jr., Posen, Poland.

After having briefly reviewed the literature on the use of typhoid vaccine in ophthalmology, the author describes his own researches and conclusions based on the treatment of 50 patients with this vaccine. The author describes in detail the technic of the treatment necessary to obtain a temperature fluctuating between 39 and 40 C. (102.2 and 104 F.) or even higher. Thereupon he presents the blood pictures, including the arterial tension and the number of leukocytes. The conclusion is that the changes are due only to the fever. There does not exist the least sign of shock or of an allergic reaction after the use of heterogeneous protein. Therapy with typhoid vaccine is, in its strict sense, pyretotherapy.

The following are the final conclusions:

1. Typhoid vaccine induces a high fever.

2. The ratio between the dose of typhoid vaccine and the temperature depends on the discretion of the physician.

3. The intravenous injection of the typhoid vaccine several times with an interval of several months between injections never induces shock.

4. The value of pyretotherapy with typhoid vaccine is considerable. The author recommends this as the fundamental and principal treatment in cases of acute uveitis, especially of the sympathetic type; in cases of retinitis or ophthalmia of unknown origin; in cases of gonorrhea, and in cases of traumatic iritis. In many other infectious diseases of the eyes, especially those produced by focal infections, typhoid vaccine acts as a strengthening therapy, accelerating the process of recovery.

Third Session, May 18

Histospectography and Microincineration of the Normal and Pathologic Crystalline Lenses and Especially of Black Cataract. H. Tillé, P. Pillet and G. Busnel, Paris.

The application of histospectography and microincineration, perfected by Policard, to the histochemistry of crystalline lenses, including all those from the normal lens to the lens with black cataract, showed the following facts:

1. There was absence of iron, copper and zinc in appreciable quantities. A minute trace of iron was found in the normal crystalline lens, in the white senile cataract and in the amber-colored senile cataract.

2. There was a clear presence of copper, not yet pointed out by various authors, in 2 cases of black cataract. The copper had its site in the nucleus.

3. The presence of iron was demonstrated in the black cataracts, with topographic peripheral predominance.

4. All lenses showed an absence of zinc.

5. Some rare melanophores were found in the black cataract of adults.

6. The results of tests for pigmentation of the red blood cells were negative; the study of the hematoporphyrins with the microspectroscope and the microfluoroscope did not reveal the existence of these pigments in any case.

Original Technics for Removal of the Crystalline Cataract (Lantern Slides). Henri Lagrange and Jean Goulesque, Paris.

Films were shown demonstrating how by the combined action of the instrument used to take hold of the crystalline lens (a cupping glass or a forceps) and an instrument for counterpressure to be applied below the cornea (a *butoir*) it is possible with a cupping glass or with a forceps to break the zonula at the level of its lenticular but not its ciliary attachment and to remove the lens with the Henri Lagrange forceps without recourse to a version capable of tearing the hyaloid membrane. Two procedures for the protection of the wide keratotomy (systematic conjunctival covering or a conjunctival bridge, with a wide flap) were also demonstrated.

Three Safe Methods of Facilitating Total Extraction of Cataract (Lantern Slides). C. Dejean, Montpellier.

The total extraction of cataract is facilitated when the patient looks down. This position is obtained by retrobulbar injection of procaine hydrochloride directed toward the superior rectus muscle at the union of its middle third with its posterior third. It is there, according to the author's investigations, that the ramifications of the motor nerve touch the muscle. The same injection can reach the ciliary ganglion and the ciliary nerves, abolishing sensation in the eyeball.

The danger of loss of vitreous in the course of the operation will be averted by hypotonia of the eyeball. This hypotonia can be achieved by retrobulbar injection. However, there are modifications in the time and individual variations. The study of these variations allows one to form the conclusion that surgical intervention ought to be begun ten minutes after the injection. Nine times out of ten this is the moment of election.

Finally, the suture of the cornea with the Gomez-Marques stitch, slightly modified, adds still more to the safety of this brilliant operation.

The Value of Total Extraction of the Crystalline Lens. P. Bonnet and L. Paufique, Lyon.

The authors discuss the advantages of total extraction of the crystalline lens in connection with 2,000 operations.

1. In cases of uncomplicated senile cataract, total extraction offers the advantage of permitting operation from the time that visual discomfort hinders reading. It offers an infinitely better functional result.

The advantages of the operation are not counterbalanced by greater risks. If the operation requires more minute precautions, a more delicate technic and a more rigorous supervision, total extraction does not, on the other hand, lead to more risks in the course of the operation or to subsequent special complications. Complications, such as detachment of the retina and delayed disturbance of the vitreous, call attention to pathologic cataracts.

2. For pathologic cataracts (cataracts associated with high myopia, subluxated cataracts, black cataracts, cataracts with descemetitis and cataracts of diabetic persons) the results obtained by total extraction are better, because the operation guards against postoperative ciliary reaction.

3. For unilateral cataract, for the same reason, it is the operation of choice.

New Series of Results of Total Extraction of Cataract: Prime Importance of Preoperative and Postoperative Care (Lantern Slides). R. de Saint-Martin, Toulouse.

The results obtained with 217 cataract extractions performed serially are reported. The patients were reexamined at least once after six months. The author compares these results with those obtained with 643 total extractions, the report of which was published in the transactions of this society in 1935 (Arch. Ophth. 14: 1044 [Dec.] 1935) to show their agreement and constancy.

Total accurate extractions have succeeded, respectively, in 91.56 and in 74.78 per cent of the cases in the first and second series. Superior visual acuity, of 5/10, has occurred in 82.49 per cent of the new series and in 84.94 per cent of the old series. Acuity has improved with time (from six months to two years and more) in 86.4 per cent of the new series and in 84.94 per cent of the old series. It has diminished in 2.32 per cent of the cases of the new series and in 8.43 per cent of the cases of the old series. The accidents for which certain critics make the method particularly responsible and which according to them are sufficient to discredit it are: detachment of the retina and progressive and late disturbance of the vitreous, which occurred in the total of 860 extractions, retinal detachment occurring in 1.04 per cent, and disturbance of the vitreous in 5.23 per cent. (The last-mentioned occurrence has its source most often in former iridochoroiditis and myopic flakes.)

Results such as these, identical with those published for more than ten years by the best European and American surgeons, are sufficient to establish the superiority of total extraction. Good results cannot be obtained except by the strict observance of preoperative and postoperative precautions, which the author enumerates and which concern, respectively, the digestive, the circulatory and the nervous system.

Systematic Conjunctival Covering in the Operation for Complicated Cataracts. H. Villard, Montpellier.

The author advises that the classic operation for removal of cataract be completed by entirely covering the corneal wound with conjunctiva

in all cases of cataract in which failure is particularly to be dreaded because of abnormal septic conditions.

The ideal protection of the wound by the mucous membrane of the conjunctiva permits the saving of eyes which without this precaution seem doomed to an almost fatal infection. The excellent results obtained in those operated on urged him to employ a similar precaution in cases in which a loss of vitreous was almost certain (cases of luxated or subluxated cataracts and secondary cataracts subjected to removal). Here, too, the results were remarkable.

Ocular Tension, Glaucoma and Hypophysis. P. Jeandelize, P. L. Drouet, C. Thomas and Bardelli, Nancy.

The authors draw the following conclusions:

1. There may be coexistence of ocular hypertension and certain hypophysial diseases.

2. Certain diseases of the pituitary gland of the hyperfunctioning type (acromegaly) or certain condition such as pregnancy, in the course of which the hypophysis undergoes important modifications, engender ocular hypertension.

3. Hypophysial extracts seem to have a hypotensive effect in cases of glaucoma.

4. The experimental data confirm the hypotensive effect on the eyeball of the injection of posterior pituitary extract and the hypertensive effect of hypophysectomy.

The facts do not permit further conclusions. New researches will be necessary to specify the constancy of the hypotensive effect of the extract of the posterior lobe, the least concentration to be used to obtain a certain effect, the mechanism of this hypotension and the clinical types of glaucoma in which therapeutic indication will seem most effective.

Attempted Treatment of Chronic Glaucoma Through Acidification of the Vitreous Body. E. Redslob, Strasbourg.

The author criticizes therapeutic attempts to diminish glaucomatous hypertension by lowering the p_H of the vitreous. He bases his stand on experiments made in collaboration with Reiss in vitro and on the rabbit. The experiments showed, in fact, that a slight lowering of the p_H of the vitreous considerably diminishes its state of distention.

He uses, as previously, a 0.7 per cent solution of phosphoric acid. This solution is perfectly tolerated by the vitreous and the entire eyeball. The same holds good for a 2 per cent solution.

The author reports his observations on 6 cases of absolute glaucoma in which this treatment was used. In all the cases there was an appreciable diminution of intraocular tonus. In 3 cases it was transitory, persisting for only about fifteen days. These half-measures were observed only in persons whose general condition was strongly affected. In the other 3 cases the effect of acidification of the vitreous body was satisfactory, whereas previous operations combined with an intensive treatment with miotics had failed. After one or two injections of acid, the tonus returned to normal and remained there for several months.

What is curious is that the miotics, without effect before the injection, had an undeniable effect after the injection.

Attempts will be made in cases of less advanced glaucoma.

MELANOCARCINOMA OF THE EYE. H. MOUTINHO, Lisbon, Portugal.

The author made an anatomicoclinical study of 9 cases of melanocarcinoma of the eyeball. Three of the growths were epibulbar, and 1 was in the iris, 1 in the ciliary body and 4 in the choroid. He establishes the preexistence of nevi in the external tumors and the coexistence of the pigmented spots of former chorioretinitis in the tumors of the choroid and accepts as the origin of the malignant melanomas a more or less apparent pigmented spot; hence the designation of melanocarcinoma to embrace the nevocarcinomas and the tumors of the choroid the nevic origin of which is not always demonstrable.

Concerning the origin of the pigment which always exists in a more or less apparent fashion, even in the leukosarcomas, the author demonstrated the endocellular formation of melanin in the culture of melanosarcomas which he undertook.

As to the histologic nature and unity of melanocarcinoma in their varied structure, the author makes a comparison between the various verified histologic aspects and the various zones of a conjunctival nevus in growth, as Masson had done for cutaneous nevocarcinomas, thus admitting the original unity of the melanomas in the epithelial melanoblast of the skin, of the conjunctiva or of the pigmentary epithelium of the retina.

Fourth Session, May 19

RATE OF BLOOD FLOW IN THE RETINAL VESSELS. M. FRITZ, Brussels Belgium.

The simultaneous observation of the vascular calibers and of the flow of blood by the technic of the artificially produced venous granular current makes possible the appraisal of the rate of blood flow at the level of the retinal vessels.

The normal rate is that which corresponds to an arterial or venous caliber of the retina of 0.1 mm. in diameter and to a flow of blood that allows the granular venous current to appear at seven tenths of arterial collapse.

A reduced vascular caliber associated with an exaggerated flow of blood indicates an abnormally fast rate. On the contrary, an exaggerated vascular caliber and a reduced rate of flow indicates a decrease of blood and predispose to thrombosis.

The rate of blood flow at the level of the capillaries can equally be ascertained to a certain degree by the simultaneous appraisal of the flow of blood and the condition of capillary permeability, indirectly appraised by the degree of venous pulsation and the intensity of the coloration of the papilla.

Angioid Streaks of the Retina, the Red Marks of the Vitreous Lamella of the Choroid: Their Relation to Pseudoxanthoma Elasticum of the Skin and to Vascular Degeneration of the Choroid; Progressive Changes of the Macula (Lantern Slides). P. Bonnet, Lyon.

The author reports 2 cases of angioid streaks of the retina. He describes the morphologic aspect of the streaks and recalls the changes in the macula and their development, referring to the articles which he published in the *Archives d'ophtalmologie* (**50**: 721 [Nov.] 1933; **52**: 225 [April] 1935).

He points out the relation of angioid streaks to pseudoxanthoma elasticum of the skin and to the degeneration of the vascular plexus of the choroid.

He believes that retinal angioid streaks correspond to cracks and red marks of the vitreous lamella of the choroid.

Cirsoid Aneurysm of the Retina (Racemose Aneurysm): Its Relations to Cirsoid Aneurysm of the Face and Cirsoid Aneurysm of the Brain. P. Bonnet, J. Dechaume and E. Bland, Lyon.

The authors, basing their opinions on two observations, call attention in *Le Journal de médecine de Lyon* of March 20, 1937 (page 165) to an anatomicoclinical complex characterized by the coexistence of retinal cirsoid aneurysm with facial cirsoid aneurysm and an intracranial cirsoid aneurysm.

A closely allied complex of retinal angiomatosis (von Hippel's disease) associated with angiomatous cysts of the brain (Lindau's disease) is considered.

The authors think that a visual disturbance, if it exists, should draw attention to the intracranial lesion.

They formulate the following conclusions:

1. In the presence of retinal cirsoid aneurysm, accidentally observed, the existence of cirsoid aneurysm of the face should be systematically looked for. A neurologic examination should be made, directed toward the search for a cirsoid aneurysm of the brain.

2. The presence of any vascular malformation of the face should lead to an ophthalmologic and a neurologic examination.

3. In the case of a cerebral disease, the ophthalmologist, examining the eyeground, should bear in mind the existence of cirsoid aneurysm of the retina.

Thrombosis of the Central Vein of the Retina. H. Coppez and A. Fritz, Brussels, Belgium.

The authors analyze 2 cases of thrombosis of the central vein of the retina, in which the ophthalmoscopic aspects were identical but the mechanisms of development of the condition were essentially different.

In the first case there was generalized hypertension; the tension of the brachial artery varied from 10 to 19.5 and that of the retinal artery from 75 to 170. The pressure of the central retinal vein was 80 mm. of mercury.

In the second case there was no general hypertension; the tension of the brachial artery varied from 8.5 to 13.5, but, on the other hand, there was marked hypertension of the retinal artery, from 25 to 85. The pressure of the central retinal vein was 45 mm. of mercury.

In the first case the mechanical factors played the main role. Hypotensive and vasodilative treatment was indicated.

In the second case the mechanical factors played only a subordinate role, the hematologic and vascular factors being in the foreground. Treatment should be anticoagulative and antiseptic. The use of vasodilators would be harmful.

The prognosis was unfavorable in the first case and favorable in the second case.

Retinal Cyst (Lantern Slides). P. Veil, Paris; L. Guillaumat, Paris, and L. Petrignani, Beauvais.

The authors report the observation of a retinal cyst the evolution of which could be followed ophthalmoscopically from its formation to its rupture. The patient was a woman aged 30, afflicted in one eye with hypertensive iridocyclitis and in the other eye with descemetitis and excavation of the optic disk, the retina being normal without any cystic formation. The inferotemporal cyst of the left eye, which was emmetropic, appeared without traumatism. The cyst was globular and had smooth walls and a clearly marked contour. It was translucent and fixed and differed in appearance from a retinal pocket due to detachment. Its progress to rupture in the vitreous proves its cystic nature. Rupture did not bring about retinal detachment. In fact, the latter is considered by Wevé to be the result of such cysts.

Relation of Detachment of the Retina to Circulatory Disturbances of the Choroid (Lantern Slides). E. Redslob and J. Nordmann, Strasbourg.

The authors report a case of orbital osteoma in which the growth had produced an enormous stasis of the choroidal veins. At the precise point where dilatation of the veins attains its maximum there were detachment of the retina and polycystic retinomalacia.

Observations made on the eyeballs of dogs in which a vortex vein had been ligated were also reported. The stasis of the choroidal veins was extensive and led to retinal and choroidal detachment and to degeneration of the retina.

It has thus been shown that in a previously healthy eye congestion of choroidal veins can cause cystic degeneration of the retina and detachment. Insufficiency of arterial circulation seems to lead to the same result, so that the huge majority of idiopathic or secondary detachments of the retina should be related to disturbances of choroidal circulation.

Oxycephaly and Atrophy of the Optic Nerve (Lantern Slides). Pierre Gorse and Louis Calmettes, Toulouse.

In connection with a case of oxycephaly the authors call attention to the various pathogenic factors invoked to explain the atrophy of the

optic nerve usually observed in such patients. The atrophy in cases of oxycephaly is almost always of the postneuritic type, which there is a tendency to consider as due to papillary stasis, a consequence of intracranial hypertension.

In the case reported the atrophy was of the primary type. The authors admit the undeniable influence of cranial hypertension, but give an important role to the other possible causes, such as the compression and elongation of the optic nerve within the cranium or the optic foramen and even its toxic-infectious effect.

They think that even in a case of postneuritic atrophy in which the role of papillary stasis appears primary one should not set aside the other factors.

COMPARISON OF THE HISTOLOGIC PICTURE OF CERTAIN ORBITAL TUMORS OF LACRIMAL ORIGIN WITH THAT OF THE SO-CALLED "MIXED" TUMORS OF THE SALIVARY GLANDS (Lantern Slides). H. TILLÉ and J. LEROUX-ROBERT, Paris.

The authors describe 15 glandular epitheliomas of the orbit, selected from 45 primary tumors of the orbit classified up to the present time as epitheliomas, fibroblastic sarcomas, round cell sarcomas, endotheliomas and, above all, cylindromas. They classify them anew, taking inspiration from the labors of Leroux on the so-called "mixed" tumors. The lacrimal glands, the aberrant glands scattered in the orbit and the conjunctiva form a part of the same regional system as the organic or submucous pituitary and salivary glands. Histologically, the difference in the structure of the tumors considered is explained, on the one hand, by the differentiation and lack of differentiation and the metaplasia of epithelial elements and, on the other hand, by the epithelioconjunctival interaction and the inversion of secretory polarity.

In short, among the 45 primary tumors of the orbit, they found 5 acinous forms, 8 acinocanalicular forms, 1 vegetative canalicular form and 1 canalicular form with eosinophilic muciparous cells. The pure acinous forms, and especially the atypical and deviating forms, seem to them to have a more unfavorable prognosis than the acinocanalicular forms, the latter having a prognosis similar to that of the so-called "mixed" tumors. The group of glandular epitheliomas described by the authors is to them by far the most frequent and important of the primary tumors of the orbit.

HEXAMETHYLENETETRAMINE (METHENAMINE) IN OCULAR THERAPY. L. GRELAULT, Paris.

Grelault reports the results obtained with injections of hexamethylenetetramine (methenamine) into the polar or limbic region combined with instillations or baths of the same substance. Hexamethylenetetramine, by its antivirus properties and its great diffusibility, which permits it to penetrate impervious systems (cerebrospinal cavity and ocular cavity), is, according to the author, a valuable agent in ophthalmologic therapy. Its use is indicated for all the chorioretinal diseases, iritis, iridochoroiditis, lymphocytosis of the vitreous body, episcleritis, corneal

lesions, virus diseases, herpes zoster and trachoma. (The treatment is given in cases of trachoma by a special technic.)

Grelault believes hexamethylenetetramine to be a therapeutic agent of the first order, worthy of the whole attention of ophthalmologists, its curative effect being achieved by stages of regular and important improvement.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

EDWARD STIEREN, M.D., *President*

Oct. 24, 1938

GEORGE H. SHUMAN, M.D., *Secretary*

LOUIS STATTI, M.D., *Reporter*

DIFFUSE ORBITAL OSTEOMA: REPORT OF A CASE. DR. ADOLPH KREBS.

Miss R. H., aged 35, was seen on Jan. 7, 1936, because of protrusion of the right eye of two weeks' duration. There had been no subjective symptoms, and she had had no illness. The exophthalmometer showed the right eye to measure 23 mm. and the left eye, 19 mm. The proptosis of the right eye increased to 29 mm. within a few months. Roentgenographic study by Dr. G. W. Grier on January 15 showed extensive thickening of bone involving the roof and the external wall of the right orbit. The process extended backward and involved the floor of the frontal fossa. From the appearance of the lesion, he believed it to be an osteoma. The orbit was considerably contracted by the extensive overgrowth of bone. Roentgenographic examination on August 5 did not show any change.

At my request, the patient consulted Dr. Walter E. Dandy, who after further roentgenographic study performed an operation for the removal of a diffuse orbital osteoma on April 9, 1937. I next saw her on October 28. The right eye was still proptosed, and it pulsated synchronously with the heart beat. Exophthalmometric measurements were 29 mm. for the right eye and 20 mm. for the left eye. There was diplopia when the patient looked up or down. The media were still clear, and the fundi were normal. Vision of the right eye was 7/12—. On Jan. 6, 1938, it was 7/30. The media were still clear, and the fundi were normal. The visual field of the right eye did not show any peripheral contraction or enlargement of the blindspot, but there was a relative central scotoma for colors.

A second operation was performed by Dr. Dandy in March 1938. He found nothing grossly to explain the return of the exophthalmos, but the tissue removed showed an infiltrating tumor. He found it to be one of the malignant types of dural endothelioma, the malignant nature making it most unusual. His opinion was that the prognosis was hopeless, and he stated that it was the only orbital tumor of this type that he had had.

Malignant Melanoma of the Ciliary Body: Report of a Case. Dr. J. Clyde Markel.

A white man aged 62, a painter, was first seen on June 6, 1937, with a history of failing vision in the right eye for four months. His health always has been good, though a number of years before he had to give up painting on account of painter's colic and became a brakeman on a railroad.

There was a mature cataract in the right eye, though perception and projection of light were good. The right pupil was slightly oval but active to light. Vision of the left eye equaled 20/40, with improvement to 20/20 with a low correction. Lenses were prescribed, and the patient was told to report in the fall.

Examination of the eyes on November 16 showed a leash of dilated vessels in the conjunctiva at the lower nasal limbus. At this same area there was a small linear black mass 2 by 0.5 mm., causing a slight fold in the iris, with fine new vessels over the surface. Transmission of light over this area was poor on transillumination. Intraocular tension was not raised. A diagnosis of melanosarcoma of the choroid was made, and immediate enucleation was urged. Dr. Curry concurred in the diagnosis. The patient decided to think it over and did not return until December 7, when the mass appeared to be slightly larger, and there was some pain. Simple enucleation was done on December 16, avertin being used for anesthetization.

According to the pathologic report, made by Capt. Elbert Decoursey, of the Army Medical Museum, the tumor consisted of an irregularly pigmented mass measuring about 1.5 mm. in diameter and extending from the ciliary body to the equator. Microscopic examination showed the tumor to extend almost to the angle of the anterior chamber and apparently to originate in the ciliary body. It had grown inward to press on the lens and invade the retina, causing detachment. The growth was composed of closely packed, moderately small cells with vesicular nuclei, prominent nucleoli and a moderate amount of cytoplasm. Pigment generally appeared only in large masses around the vascular portions. There were occasional small areas of necrosis, with little or no round cell infiltration. According to the Callender-Wilder classification, the tumor consisted of somewhat less than 50 per cent argentophilic fibers. The uninvolved ciliary processes showed some hyalinization. The lamina cribrosa was not depressed. The lens showed irregular cortical degeneration, and where it approximated the tumor exhibited a thickened capsule. The nucleus of the lens had been pushed out of line. The diagnosis was malignant melanoma of the ciliary body of the small epithelioid cell type, and traumatic cortical and equatorial capsular cataract secondary to pressure of the tumor mass.

Dystrophia Mesodermalis Congentia, Typus Marfan: Report of a Case. Dr. Edward Stieren.

A woman aged 35 had strongly contracted pupils, iridodonesis and dislocation of both crystalline lenses upward and inward. The lenses were opaque, the patient being able to see in the area above the dislocation with + 12 D. lenses. The pupils would not dilate with any mydriatic. The patient was abnormally tall, 5 feet and 11 inches (180

cm.), with long narrow feet and hands. Her emaciated appearance was due to a universal absence of subcutaneous fat, and the breasts were entirely flat. A diagnosis of arachnodactylia was made from the group of symptoms presented by the patient and first described by Marfan in 1896, known as Marfan's syndrome.

HERPETIC IRITIS WITH NEUROPATHIC KERATITIS: REPORT OF A CASE. DR. GEORGE H. SHUMAN.

Dr. W., aged 73, was first seen on Feb. 3, 1937. He had not been well for several months. In the preceding January he had been confined to bed with so-called "flu," severe bronchitis and a profuse discharge from the nose. The highest temperature recorded was 101 F. In the early morning of February 2 he was aroused by a severe pain in the right eye accompanied by a feeling of scratchiness. When he was seen on the morning of February 3, he had a severe ocular pain; practically the entire cornea, except for a narrow margin at the periphery, was covered with rather gross filaments and shreds of epithelium, yet none of the involved area stained well with fluorescein, a finding which is not unusual in the so-called dystrophic disturbances of the cornea. Biomicroscopic examination did not give any additional information of value on the superficial aspect of the cornea, but it did disclose numerous large folds in Descemet's membrane, greatly increased albumin content of the aqueous, which was literally swarming with inflammatory cells, and absence of the convection current. Scattered over the iris were small, discrete hemorrhages, situated in the stroma. It was not possible to study these hemorrhages minutely, because the clouded cornea and turbid aqueous interfered with visibility. There was neither hyphema nor hypopyon.

Based on the age of the patient (lowered vitality), the history of an antecedent febrile infection of the upper part of the respiratory tract, the severity of the iritis and the presence of interstromal hemorrhages in the iris, together with the character of the corneal involvement, a diagnosis of herpetic iritis with a complicating neuropathic keratitis was made.

When the patient was seen one week later, there was no abatement in the severity of the iritis. The appearance of the cornea had changed from that of a filamentous keratitis to an appearance characteristic of neuroparalytic keratitis; that is, there was a simple and more or less complete denudation of the epithelium over the central region involving roughly about two thirds of the corneal surface. The patient refused hospitalization until he later became irrational, partly from exhaustion due to pain and loss of sleep and possibly also from the too frequent or careless use of hyoscyamine in the eye at home. He objected to the use of atropine because of a pronounced general idiosyncrasy to it. Under rest and general care, local use of heat and bland cleansing irrigations and ointments, the cornea regained its epithelial covering in about seven weeks. The inferior half of the involved cornea became deeply infiltrated, and the central part at one time took on a yellowish appearance. A dense central scar remained to obstruct vision in the lower half of the pupillary area. The lower edge of the pupil was also obstructed by a dense synechia formed of exudate, which was also

spread over the anterior lens capsule. The biomicroscope showed a layer of dense infiltration just anterior to Descemet's membrane similar to that which persists after syphilitic interstitial keratitis. Corrected vision was 20/70. Intramuscular injections of posterior pituitary were given, with apparent benefit, for recurrent attacks of severe neuralgic pain. No other of the usual causes of iritis was found.

BILATERAL DEHISCENCE OF THE ANTERIOR LENS CAPSULE: REPORT OF A CASE. DR. JOHN B. MCMURRAY.

Mr. E. H., aged 50, a steel worker for many years, was hospitalized on Feb. 22, 1937, because of a severe zoster ophthalmicus on the right side. He remained in the hospital for fifty-six days. He consulted me on November 27 on account of severe almost continuous pain over and in the right eye. This pain was undoubtedly due to the attack of zoster ophthalmicus. In examining the eye, however, I found that he had an exfoliation of the anterior capsule of the right lens. This was an octagonal-shaped leaf of exfoliation, with its base toward the temporal side. In the left eye he had the same condition of the anterior capsule. There was a large leaf that was turned to the nasal side. The vision in the right eye was reduced to 20/200 and could not be improved. In the left eye the vision was 20/50, and with a + 0.75 sphere it was improved to 20/40. The tension with the Schiötz tonometer was 15 mm. in either eye. The visual fields were normal. The last time I examined the patient, on Oct. 17, 1938, I found the exfoliation to be just about the same as at the former examination. The tension and the visual fields were normal. The vision was the same as at the first examination.

Book Reviews

Allergische Augenerkrankungen. By Prof. Arnold Loewenstein, Prague. Price, 4.10 marks. Pp. 72. Basel: S. Karger, 1938.

In this treatise, issued as a supplement to the current volume of the *Zeitschrift für Augenheilkunde* (Kuhnt and von Michel), an introductory chapter is devoted to a general consideration of the factors, offensive and defensive, which play a part in allergic reactions and to definition of the terms and concepts applied. The author then considers, in turn, ophthalmic allergic reactions due to the instillation into the conjunctival sac of such medicaments as atropine, scopolamine and physostigmine and certain excipient vehicles, such as petrolatum and sesame oil; to matter unintentionally involving the skin of the lids or the conjunctiva such as toxic (sic) menstrual, fish or cat's blood, dust, dander, feathers and animal, plant and flower "hairs," and to substances which have a general injurious effect in addition to a local reaction, such as substances which give rise to industrial idiosyncrasies, cutaneous allergies and allied conditions. There is a chapter on the determination of the allergen by instillations of the antigen into the conjunctival sac (Dunbar, by means of the nasal test [Urbach; Gutmann]) and by cutaneous (the Jadassohn-Bloch patch test) and intracutaneous tests. A number of clinical forms are described and discussed, including the ocular complications of hay fever, its etiology and treatment, vernal catarrh, experimental anaphylactic keratitis, familial corneal degeneration, the "moon blindness" of horses and allergic inflammation of the sclera, iris and ciliary body. The author is not convinced of the allergic nature of sympathetic ophthalmia and endophthalmitis phacoanaphylactica.

PERCY FRIDENBERG.

Die Veraetzungen des Auges. By Dr. Oskar Thies, Dessau. Price, brochure, 4.60 marks. Pp. 103, with 36 illustrations. Stuttgart: Ferdinand Enke, 1938.

In a thoroughly documented and copiously illustrated monograph, which appears as a supplement to the *Klinische Monatsblätter für Augenheilkunde*, the author considers at length new paths in the interpretation and treatment of those chemical injuries of the eyeball and/or lids which in English medical parlance are summed up as "burns." Thies gives in detail the pathologic factors, stages, clinical course, complications and sequelae in corrosions by acids and alkalis as well as by ammonia and lime and such varied chemical products as ethereal oil, aniline dyes, alcohol, combat gases and metallic compounds. There is an excellent chapter on first aid and immediate treatment by irrigation, neutralization and protection and the early surgical restoration by means of skin and/or buccal mucous membrane grafts. The work is thorough, like most German treatises, and like them, it practically ignores the publications of American and English ophthalmic surgeons.

PERCY FRIDENBERG.

Zur Pathologischen Anatomie und Therapie des Trachoms. By Prof. A. Birch-Hirschfeld, Königsberg. Price, 4.10 marks. Pp. 42, with 18 illustrations. Basel: S. Karger, 1938.

The author, noting the surprising multiplicity of clinical and histopathologic pictures in the course of trachoma, has limited himself mainly to the study and interpretation of changes in the tarsus and the tarsal conjunctiva. He considers the tissue manifestations of the later stages to be at least as distinctive and important as, for example, the various cell forms found in the infiltrated regions, the trachoma follicle or changes in the conjunctival epithelium. Thorough microscopic study of 153 excised tarsi showed in spite of a marked variation in the intensity and localization of the inflammatory and infiltrative processes that there are certain definite and characteristic changes and, essentially, correspondence in later stages. A knowledge of these factors which determine in a large measure the final condition of the lids and to some degree that of the globe is of practical significance, as they point directly toward the appropriate treatment, medical and mechanical, of trachoma in its initial stages and to early combined excision of the tarsus, when this structure has become involved. The monograph is published as a supplement to the *Zeitschrift für Augenheilkunde* (Kuhnt and von Michel).

PERCY FRIDENBERG.

The Vitamins and Their Clinical Application. By W. Stepp, J. Kühnau and H. Schroeder. Translated by Herman A. H. Bouman, M.D. Price, $4.50. Pp. 173, with a separate index. Milwaukee: The Vitamin Products Company, 1938.

This manual was published in Germany late in 1936. The translation appeared in 1938 with an appendix of 53 pages, which, according to the publishers, brings the edition up to date until October 1937.

The first 120 pages (the translation) discusses briefly for each vitamin its history, chemistry, determination, occurrence, physiology, manifestations of deficiency, supposed mode of action and, in some instances, commercial preparations and dosage. The authors have assembled a mass of information in a few pages. If one remembers that this portion of the book covers the field only up to 1937, it can be said that it furnishes a compact body of data and an adequate bibliography.

The appendix, on the other hand, seems superfluous. Fifteen pages are devoted to outlines of the apparent function, possible results of deficiency and results of absence of each vitamin.

The separate bibliography of the appendix is imposing with over 680 references. However, 337 of these refer to the same 12 sources, so that the bibliography is at least twice as long as is necessary. Five of these 12 sources are textbooks which are, after all, secondary or tertiary sources of information.

WALTER F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, 81 Edmund St., Birmingham, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1.
Time: April 20-22, 1939.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Hon. Secretary-Treasurer: Dr. F. A. Anderson, 12 St. John's Hill, Shrewsbury, England.
Time: July 6-8, 1939.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian Str. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel-Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel-Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby Str., Tel-Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik Str., Tel-Aviv, Palestine.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung, China.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

Canadian Ophthalmological Society

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. L. Goss, Cobb Bldg., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: Capitol Life Bldg., Denver. Time: 7:30 p. m., third Saturday of each month, October to May, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. J. M. Lilly, 302 Old St., Fayetteville,
Secretary-Treasurer: Dr. Frank C. Smith, 106 W. 7th St., Charlotte.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.
Place: Fargo. Time: May 1939.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. Kate Savage Zerfoss, 165–8th Ave., N., Nashville.
Secretary-Treasurer: Dr. W. D. Stinson, 805 Medical Arts Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. T. E. Fuller, 100 W. Board St., Texarkana.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. J. Mason Baird, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. Alton V. Hallum, 478 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St. Time: Second Friday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. Clifford B. Walker, 427 W. 5th St., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Pl., Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. N. H. Turner, 200 E. Franklin St., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. R. E. Elliott, 78 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. Raphael Farber, 280 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Roy E. Mason, Frisco Bldg., St. Louis.
Secretary: Dr. Leslie Charles Drews, 508 N. Grand Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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COMMON WART AS AN ETIOLOGIC FACTOR IN CERTAIN CASES OF CONJUNCTIVITIS AND KERATITIS

A. DE RÖTTH, M.D.
CHICAGO

The most frequent ocular disease, conjunctivitis, may puzzle the expert and cause great worry to the patient. The lesion usually heals in several days, but it may resist the same type of treatment for several weeks and cause complications. This difference in response is due principally to the different etiologic factors. Thygeson [1] has presented a thorough enumeration of the different kinds of treatment for conjunctivitis, and from the practical point of view I have high esteem for his paper, as he considers the treatment on the basis of causation.

In 1933 and 1935 I [2] described 2 cases of conjunctivitis and 3 cases of keratitis due to common warts of the margin of the lid. My first observation dates from 1930. Up to the time of writing I have observed 10 such cases, including the first series of 5 cases. Having seen about 32,000 new patients with ocular conditions during these eight years I place the frequency of the occurrence of this etiologic factor in Hungary at about once in every 3,200 cases of ocular disease. Since my first papers on this subject, Vito [3] in 1936 described 3 cases of follicular conjunctivitis due to common wart, including 1 case due to a juvenile wart of the eyelid. The excision of the wart was followed by complete recovery. The instillation of the ground verruca into the other eye of the patient resulted in 1 case in conjunctivitis, but when the material was instilled into the conjunctiva of a normal eye it caused only slight hyperemia.

From the State Eye Hospital (Prof. Dr. J. Imre), Budapest, Hungary.

1. Thygeson, P.: The Treatment of Conjunctivitis, Arch. Ophth. **19**:586 (April) 1938.

2. Rötth, A.: Die Bedeutung der Verruca des Lidrandes in der Aetiologie gewisser Bindehaut- und Hornhautentzündungen, Klin. Monatsbl. f. Augenh. **91**: 196, 1933; L'ultravirus de la verrue vulgaire facteur étiologique de certaines conjunctivitis et kératitis, in Deliberationes Congressus Dermatologorum internationalis, Leipzig, Johann Ambrosius Barth, 1936, vol. 2, p. 262.

3. Vito, P.: Sulla congiuntivite da verruca del bordo libero delle palpebre, Boll. d'ocul. **15**:627, 1936.

REPORT OF CASES

CASE 1.—Miss T. I., aged 31, suffered from hemoptysis a number of years before the present complaint. At the time she was examined for an ocular condition she was subfebrile. Roentgenograms showed enlarged and ramified hilar shadows and a veiled apex. The right eye had been irritated for two and a half months; from time to time there were severe pains and photophobia. The patient was treated with a 1 per cent solution of silver nitrate, mild mercurous chloride and atropine. On Aug. 7, 1930, the right eye ached, and there were itching and photophobia. In the center of the cornea there was an area of fine unevenness of the epithelium 3.5 mm. in diameter, and the bulbar conjunctiva was moderately congested. On the free margin of the upper lid several millimeters from the tear point there was a narrow, lobate common wart, 2.5 mm. in length. Therapy consisted of the application of an ointment containing 5 per cent novoform [4] and the administration of calcium orally. Recovery occurred in two days. On the night of August 31 the patient suffered from a severe pain in the right eye. Examination disclosed photophobia, lacrimation, a stippled cornea and slight infiltration at the periphery. The same ointment was again applied. On September 1 the pains became more severe. The greatest part of the corneal epithelium was found to be worn off. An ointment containing 5 per cent ethylmorphine hydrochloride was applied, and the eye was bandaged. On September 2 the corneal epithelium had regenerated, but there was a fine disciform haziness, which disappeared the next day. After the last two relapses, as I was unable to find any cause for the condition, I suspected, because of the analogy to molluscum contagiosum, that the wart on the lid was the pathogenic factor. On September 18 the wart was removed, and there have been no more relapses.

CASE 2.—Mrs. T. J., aged 38, had telangiectasis of the face. The right eye was removed because of an injury received when the patient was a child. Inflammation of the left eye began three months before this operation. The patient was treated for infiltration of the periphery of the cornea by several ophthalmologists with ointments containing ichthammol and zinc oxide; novoform; ethylmorphine hydrochloride and silver hexamethylenetetramine nitrate. A slight improvement occurred, but during the last month of treatment the condition became progressively worse and the eye painful. On March 19, 1932, there were aching, tearing and photophobia. The bulbar conjunctiva was highly injected. Temporally from the center of the cornea there were two slightly prominent areas of infiltration the size of a pinhead. A rich formation of new vertical corneal vessels was seen in the upper third of the cornea; 2 mm. from the upper corneal margin there were several small depressions, traces of corneal infiltration. The pupillary region of the cornea was moderately hazy and stippled. Under cycloplegia with atropine the pupil was moderately large; there were no posterior synechia. Vision was limited to ability to see fingers at 2 meters. On the border of the temporal and middle third of the lower lid between the lashes there was a common wart the size of a millet seed with an uneven surface. On March 20 the wart was removed, and ointments containing ichthammol and zinc oxide, and novoform were applied. On March 21 the hyperemia of the bulbar conjunctiva diminished, and the patient experienced great subjective relief. On March 24 the corneal infiltration cleared. Vision of the left eye was 5/30. On March 30 there was no irritation at the site of the infiltration but a slight unevenness. Vision was 5/15.

CASE 3.—B. J., aged 80, a thin, pale woman, had a progressive inflammation of the left eye of five weeks' duration. She had never had an ocular disease before.

4. Novoform ointment is a combination of bismuth oxide with tetrapyrocatechin.

On March 19, 1932, there was conjunctival injection of the left eye, with a moderate amount of discharge. In the periphery of the cornea there was observed a wide, strongly infiltrated ulcer extending from 9 to 5 o'clock, i. e., over two thirds of the corneal circumference. The most marked infiltration was in the upper part of the cornea and extended temporally down. Between the limbus and the ulcer there was a hazy strip 1 mm. in length which had not yet ulcerated. The portion of the cornea covered by the ulcer was hazy but transparent. Staphylococcus albus was cultured. Vision in the left eye was limited to perception of fingers at 2 meters. A large wart was present between the lashes of the lower lid, and there were two small ones between the lashes of the upper lid. Their surface was villous and moist. The right eye was normal. Therapy consisted of the application of ointments containing ethylmorphine hydrochloride, ichthammol and collargol and the use of atropine and hot compresses. On March 25 the corneal ulcer was deeper and broader; superficial blood vessels reached to the periphery of the ulcer. On March 26 the warts were excised. Histologic examination showed the picture of typical warts—long and broad papillae, the deepenings between them being filled with horny cells, and a sharp boundary of the epidermis. On March 29 no progression of the ulcer was noted. On April 4 the infiltration was diminishing in the periphery of the ulcer, and the eye was much paler. On April 8 the base of the ulcer was clean. On April 14 the ulcer epithelized, and a normal recovery followed.

Case 4.—S. S., a boy aged 13 years, for about a half a year had slight congestion of the right eye with some discharge. He was treated with "eye drops" without any improvement. On Aug. 5, 1932, between the lashes of the right upper lid, a little temporally from the middle, there was observed a small rugged wart, 2.5 mm. in length (fig. 1). The tarsal conjunctiva was a little thickened, but had an even surface. The bulbar conjunctiva was slightly injected. There was little discharge. Incubation revealed Staph. albus. The left eye was normal. On the back of the hand there was a flat common wart, 3 by 3.5 mm. in size. On August 7 the wart on the lid was removed; no other therapy was instituted. The patient was not seen for three weeks, when examination showed the conjunctiva to be pale and thin, like that of the left eye.

Case 5.—K. F., aged 44, had suffered from tearing and a discharge from the left eye for a month. The palpebral conjunctiva was moderately hyperemic and swollen but had a smooth surface. Culture of the discharge yielded Staph. albus and Bacillus xerosis. Twenty-three years before and also two years before the present complaint the patient had warts on the left upper lid. One year before she had had warts on the fingers. At the time of writing she had a common wart with a broad base on the margin of the left upper lid. The right eye was normal. The conjunctiva was treated for two weeks with different kinds of astringent drops, with no improvement. The patient was not willing to have the wart removed.

Case 6.—G. M., aged 59, had congestion of the left eye for two weeks. He used warm compresses and was treated with a 1 per cent solution of silver nitrate and drops of an astringent collyrium. On April 25, 1934, the palpebral conjunctiva of the left eye was swollen and red, and the bulbar conjunctiva was highly injected. There was a discharge both day and night. Incubation yielded Staph. albus. The skin around the external canthus was eczematous. On the margin of the lower lid between the lashes two small common warts with loosened surface were observed; one was near the external canthus, and the other was about 10 mm. from the former. The right eye was normal. Therapy consisted of the instillation of drops of a 2 per cent solution of argentamine (solution of silver phosphate

in an aqueous solution of ethylenediamine) and the application to the lids of an ointment containing novoform and zinc oxide. On April 27 the discharge was more extensive, and the lids were swollen. Drops of zinc sulfate, racemic ephedrine and epinephrine hydrochloride were administered. On April 28 the eye was painful. A little temporally from the center of the cornea there were three gray elevations the size of a pinhead. An ointment containing novoform was applied. On April 30 there was considerable discharge. On May 1 the warts were excised. Histologic examination showed typical papillary hypertrophy covered with thickened epithelium. The next day eczema of the lids developed under the dressing with considerable discharge. No dressing was applied, and novoform powder and drops of zinc sulfate, racemic ephedrine and epinephrine hydrochloride were used. On May 4 considerable improvement was noted, and there was less discharge. The same therapy was used. Five days later there was no discharge, and the conjunctivitis had healed.

Case 7.—Miss K. S., aged 39, had congestion of the left eye of three weeks' duration. She noticed a wart on the left upper lid two months before. Drops

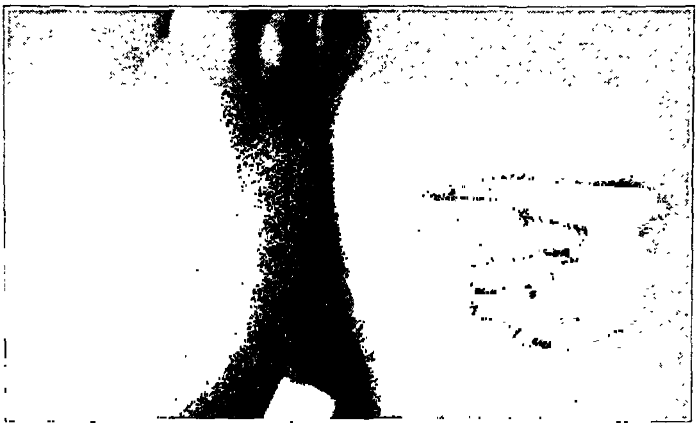

Fig. 1.—Appearance of warts in case 4.

of resorcinol were used, without results. Her maid had several warts on the hand. On May 2, 1934, the left eye was still congested, the palpebral conjunctiva was slightly thickened, the surface was smooth, and there was little discharge. Culture yielded only some colonies of Staphylococcus aureus and B. xerosis. A small wart was observed between the lashes of the upper lid near the external canthus. A little temporally from the middle there was a slightly uneven growth (wart?) several millimeters in diameter on the free margin. The right eye was normal. On May 7, as the subacute conjunctivitis did not improve with the use of drops of zinc sulfate, I excised the wart. On May 11 the conjunctiva was normal.

Case 8.—Mrs. S. J., aged 25, had blurred vision in the left eye. There was discharge, and the eye was sensitive. For five months there had been a small growth on the left lid and for five weeks one on the upper lid. A rugged hard verruca developed on the left ring finger. On June 26, 1936, examination showed papillary hypertrophy on the upper edge of the tarsus, follicles in the lower fornix and a small number of follicles in the upper fornix as well. The tarsal conjunctiva was moderately congested. In the temporal part of the lower lid and in the middle of the upper lid there were typical warts between the lashes. On

June 27 the warts were excised. On July 3 the conjunctivitis had disappeared. Two years later the patient had several warts on the fingers of the right hand.

Case 9.—I. B., aged 45, had congestion of the left eye for five days, and at the time of examination the eye was sensitive to touch. On Nov. 3, 1937, the nasal third of the conjunctiva, i. e., the semilunar fold and its surrounding region, were congested, but there was little discharge. On the upper lid, 6 mm. from the punctum lacrimale, between the lashes there was a wart the size of a pinhead, with a moist, loosened surface. Culture of the discharge revealed some B. xerosis and staphylococci. The left eye was normal. Drops of zinc sulfate and epinephrine hydrochloride were prescribed, but the removal of the wart was proposed if there was no improvement within a few days. On November 6 there was less conjunctival hyperemia and the wart was shrinking. On November 12 the shrunken, dry wart came off and the conjunctiva was normal.

Case 10.—Mrs. H. J., aged 21, had had a burning of the left eye for three months. Sometimes the eye was red. There was little discharge. Since that

Fig. 2.—Appearance of warts in case 10.

time she noted a wart on the upper margin of the lid and two weeks later, another. On July 18, 1938, examination of the left eye showed slight inflammation of the palpebral conjunctiva, a smooth surface and no follicles. Culture of the discharge yielded B. xerosis, Staph. albus and three colonies of Staph. aureus. There were a large and a small verruca between the lashes of the upper lid, one at the border of the external and middle third of the lid and the other in the middle of the lid (fig. 2). Two cilia grew through the large wart. The warts were not loosened and were covered with a fine layer of desquamated epithelium. The right eye was normal. On July 19 the wart was excised. Five days later the conjunctiva was normal.

The accompanying table shows the most important data in these cases.

The ages of the patients varied from 13 to 80 years, and the condition seemed to show no preference for age or profession. The patients included 7 females and 3 males. The disease developed eight times on

the left side and twice on the right side. The number of cases is too small to draw any conclusion.

I wish to report an additional case in which both eyes were affected.

Miss G. D., aged 55, suffered from tearing for several weeks, the use of drops being without benefit. On May 27, 1936, examination showed the palpebral conjunctiva of both eyes to be moderately congested, with a fine unevenness and little discharge, that present apparently being from the meibomian glands. There was beginning cataract in the right eye, the spokes being in the lower half of the posterior cortex. At the temporal end of the right upper lid and at the nasal end of the left upper lid there was a small common wart 2 mm. from the free margin. As the patient was living where there were many flowers in her surroundings, I did not neglect the possibility of an allergic conjunctivitis and prescribed drops of zinc sulfate, cocaine and racemic ephedrine and advised that no flowers be kept in the room. On June 12 there was no improvement. Both warts were excised. On June 16 the conjunctivitis had healed.

Data on Cases of Conjunctivitis Associated with Warts

Case No.	Sex	Age	Upper or Lower Lid; No. of Warts	Right or Left Eye	Presence of Warts Elsewhere	Duration of the Disease	Healing After Removal	Diagnosis
1	F	31	Lower lid; 1	R	....	Several relapses within 4 mo.	After removal; no relapse	Superficial punctate keratitis (?)
2	F	38	Upper lid; 1	L	....	3 mo.	4 days	Corneal infiltration
3	F	30	Lower lid, 1; upper lid, 2	L	....	5 wks.	12 days	Corneal ulcer
4	M	13	Lower lid; 1	R	Back of right hand	6 mo.	When controlled after 3 weeks found normal	Subacute catarrhal conjunctivitis
5	F	44	Lower lid; 1	L	Fingers	1 mo.	No removal	Subacute catarrhal conjunctivitis
6	M	59	Upper lid; 2	L	....	2 wks.	8 days	Acute catarrhal conjunctivitis
7	F	39	Lower lid; 1	L	On her maid's hand	3 wks.	4 days	Subacute catarrhal conjunctivitis
8	F	25	Lower lid, 1; upper lid, 1	L	On the left ring finger	5 mo.	6 days	Subacute follicular conjunctivitis
9	M	45	Lower lid, 1	L	....	A couple of days	Wart shrunken within 2 months; recovery	Subacute catarrhal conjunctivitis
10	F	21	Lower lid; 2	L	....	3 mo.	6 days	Subacute catarrhal conjunctivitis

In 6 cases there was but one wart, in 3 cases two warts and in 1 case three warts. Only the upper lid was involved in 6 cases and only the

lower lid in 2, while in the remaining 2 cases both the upper and the lower lid were involved. The warts were generally small, the bases measuring from 1 to 2 mm. and the length from 1.5 to 2.5 mm.; the surface was uneven or villous, and the warts were mostly loosened and moist. These warts should not be confused with other growths, such as small fibromas, chalazions, moles, etc., on the margin of the lid. In cases 3 and 6 histologic examination was made, the results strengthening the clinical picture. Only in cases 5 and 10 were the warts dry and covered with a fine scale of epidermis. In case 5 the wart had a broad base. The source of infection was discovered in 4 cases (4, 5, 7 and 8), once on the back of the hand and twice on the finger, and once the maid had warts. The duration of the illness was found to vary from two days to six months.

All but 1 patient was treated with the usual up-to-date medicines used for conjunctivitis or keratitis. This patient suffered only a few days from a slight conjunctivitis, and I gave him drops of zinc sulfate and epinephrine hydrochloride but proposed that the wart be removed if no improvement occurred. The wart dried up and came off in twelve days. In the meantime the conjunctivitis improved.

All the patients recovered within a few days after removal of the warts without any treatment or with treatment no different from that used before removal. One patient refused to have her wart removed; after treatment for two weeks she left the polyclinic without being cured.

The clinical picture in the majority of the cases was that of subacute conjunctivitis, consisting of moderate inflammation and slight swelling of the conjunctiva, a smooth surface and little discharge. In case 6 the picture was that of acute conjunctivitis with a badly swollen fornix. However, the patient was of the eczematous, edematous type. Several follicles were present only in case 8, though the conjunctivitis in the 3 cases reported by Vito [3] was of follicular type.

Bacteriologic examination was made in cases 3, 4, 5, 6, 7, 9 and 10. The incubation of the conjunctival discharge on blood agar and serum broth yielded Staph. albus (cases 3, 4, 6 and 10), Staph. aureus (cases 7 and 10) and B. xerosis (cases 5, 7 and 10). The simple examination of the smear in case 9 yielded staphylococci and B. xerosis. These are the usual saprophytes of the conjunctiva, though Staph. aureus is often, and Staph. albus sometimes, pathogenic. But these micro-organisms did not cause the disease in my cases, as they were found only in small numbers. In cases 3 and 10 the number of colonies of Staph. albus were numerous. It may be that the ulcer in case 3 was caused by a secondary infection. But in the two cases, just as the others, the condition healed quickly after removal of the wart without any other treatment of the conjunctival flora.

In 4 cases (1, 2, 3 and 6) keratitis of different types was observed. In the first case there was unevenness of the cornea in a circumscribed area. The patient could not be examined by the slit lamp, because of extreme photophobia and pain, but it was certain that only the epithelial layer was involved, as in punctate epithelial keratitis. It is not likely that herpes would heal in two or three days, as the condition in this case did. The possibility of phlyctenular keratitis was considered, as the patient had swollen hilus glands and was subfebrile. However, she had never had a regular phlyctenule. I observed an attack of keratitis twice, but probably they had occurred several times before. No corneal vessels developed. The definite recovery after removal of the wart strengthened the supposition that in this case a wart may have produced an ocular disease. This type of keratitis was observed in another case in association with warts, but as they were on the forehead the case is not included in this series of 10. However, it is reported here.

In V. L., a writer, aged 32, redness of the right eye developed two weeks before the reported examination. Recovery occurred in a few days. Three days before, the patient had a relapse, characterized by much itching and photophobia. Examination on April 18, 1934, showed moderate hyperemia of the palpebral conjunctiva but no discharge. Therapy consisted of the use of drops of a 2 per cent solution of argentamine and warm compresses. The left eye was normal. On April 20 an ointment of ichthammol was applied. On April 23 the conjunctiva was almost normal; no further treatment was given. On May 3 the patient awoke with severe pains in the right eye. Nasally from the center of the cornea there were three fine, slightly elevated infiltrations. An ointment containing novoform was applied. On May 4 there was no change in the patient's condition. Ointments containing ethylmorphine hydrochloride and novoform were used. There were now observed in the middle of the forehead three flat but uneven warts. The patient did not know how long he had had them. On May 5 the condition was the same. On May 6 there was conjunctival hyperemia. Drops of zinc sulfate and racemic ephedrine were instilled. On May 7 photophobia was noted. Slit lamp examination showed several filiform threads 1.5 mm. from the upper edge of the cornea. An ointment containing ethylmorphine hydrochloride was applied. On May 8 there was no change. On May 9 new filiform threads were observed toward 11 o'clock. The warts on the forehead were suggested as a possible cause of the keratitis, but the patient would not consent to their removal and did not return for treatment.

In this case the etiologic role of the warts was not obviously proved, as the patient was not willing to have the warts removed and disappeared from observation.

As the second case of corneal involvement, I mention case 6. During the observation and treatment of the patient three gray elevations the size of a pinhead appeared near the center of the cornea. These disappeared in several days. In the third case of corneal involvement (case 2) there were two large areas of infiltration between the limbus and the center of the cornea. A considerable number of new vessels and a haziness beyond the infiltration dominated the picture. This status of the eye and the condition of the face suggested keratitis due to acne rosacea.

Because of failure of the specific therapy, ichthammol ointment, and the prompt recovery after removal of the wart, this case, too, had to be accepted as one in which the wart was the etiologic factor. In the case of the most severe keratitis (case 3) the ulcer measured 2 or 3 mm. in breadth and extended over two-thirds the circumferencee of the cornea. During observation and treatment of the patient in the hospital the condition became worse. This change may have been due to the development of a row of superficial infiltrations. The age and poor health of the patient must have influenced the progress of the ulcer.

Only this last case in which there was a moderate discharge can be considered as an example of progression to a catarrhal ulcer from a row of peripheral infiltrations. In all the other cases of keratitis the condition likely began as a primary attack in the cornea. These cases represent different degrees of the same process. In cases 1 and 6 and in the case of V. L. the epithelium seemed to be directly attacked by the exogenous factor similar to that in superficial punctuate keratitis or by the finest superficial infiltrations. The condition in these cases developed suddenly with severe pains and photophobia; no vessels formed, and complete recovery occurred within several days. Thus only the epithelium could be involved. The infiltration in case 2 was more extensive and deeper than in the other cases. In case 3 other disadvantageous factors allowed this infiltration to penetrate to the deeper layers of the cornea and cause a confluent peripheral ulcer.

Because of the lack of an accompanying conjunctivitis in cases 1 and 2 and in the case of V. L., involvement of the cornea was considered to be due to the verruca virus.

All patients showed a conformity in the following characteristics: 1. The disease was unilateral. 2. One wart or several were present. 3. The superficial layers of the eye were attacked, pointing to the probability of an exogenous factor. 4. The condition resisted the usual types of treatment. 5. The disease improved after several days simply by removal of the warts. There was no improvement in case 5 during the period the patient was observed, and she refused to have the wart excised. From these facts one may draw the conclusion that in these cases there was a causal connection between the wart on the lid and the ocular disease through the filtrable virus of the common wart.

Verruca vulgaris of the margin of the lid is not an especially rare condition. According to Darier [5] it is common on the hand and fingers, but is rarer on the plantar surface of the foot, on the trunk, on the eyelids or on the hairy scalp. The condition has always been considered contagious. The infectivity was first proved by Variot [6] (1894) and Jadas-

5. Darier, J.: Précis de dermatologie, ed. 4, Paris, Masson & Cie, 1928.

6. Variot, G.: Un cas d'inoculation expérimentale des verrues de l'enfant à l'homme, J. de clin. et de thérap. inf. **2**:529, 1894.

sohn[7] (1896) and the filtrability of the virus was later proved by Ciuffo[8] (1907). The period of incubation ranges from one to five months. Lipschütz[9] found in the upper layer of the prickle cells covering the wart, but especially in the cells of the horny layer, intracellular inclusions characteristic of the disease. These inclusions are basophilic karyooikons, in the nuclei like the inclusions of herpes febrilis. They are round or oval, measure several microns in diameter, and are gram-negative; they probably are the product of the reaction of the cells to the virus.

Is there any evidence of the pathogenicity of this virus for the conjunctiva and the cornea? It goes without saying that there is no clinical identity between the wart and conjunctivitis or keratitis. The wart does not grow on these tissues; it has been observed in only a few cases growing on the caruncula. The clinical picture produced by warts does not include the presence of wartlike products on the conjunctiva or cornea. However, this is due to the different reactivity of these tissues as compared to that of the skin; also the other viruses of inclusion diseases of the skin such as molluscum contagiosum, variola, variolovaccinia, herpes febrilis and herpes zoster, all produce a different clinical picture on the skin and on the eye. Only verruca vulgaris, the etiology of which is considered identical to that of condyloma acuminatum, is not known to cause an ocular disease.

Of the inclusion diseases of the skin, the wart resembles most molluscum contagiosum. Both of these diseases belong to the group of bryocytosis, i. e., diseases in which the virus causes a tumor-like proliferation. The significance of molluscum contagiosum on the lid and its relation to conjunctivitis was emphasized first by Mütze[10] (1896) and then by Elschnig[11] (1897), H. and S. R. Gifford[12] (1921) and others, such as Cipolle, Cavara, Redslob, Goodpasture, Bardelli, Nichelatti and Kiefer. In 1931 I[13] reported a case in which these two conditions were present. The conjunctival condition in the 4 cases I have had the opportunity of

7. Jadassohn, J.: Sind die Verrucae vulgares übertragbar? Verhandl. d. deutsch. dermat. Gesellsch. (1895) **5**:497, 1896.

8. Ciuffo, G.: Innesto positivo con filtrato di verruca volgare, Gior. ital. d. mal. ven. **42**:12, 1907.

9. Lipschütz, B.: Die Einschlusskrankheiten der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1931, vol. 11, pt. 1.

10. Mütze, H.: A Contribution to the Study of Molluscum Contagiosum of the Eyelids, Arch. Ophth. **26**:15, 1897; Arch. f. Augenh. **33**:302, 1896.

11. Elschnig, A.: Molluscum contagiosum und Conjunctivitis follicularis, Wien. klin. Wchnschr. **10**:943, 1897.

12. Gifford, H., and Gifford, S. R.: Molluscum Conjunctivitis, Arch. Ophth. **50**:227, 1921.

13. Rötth, A.: Molluskum-Konjunktivitis, Klin. Monatsbl. f. Augenh. **86**:543, 1931.

seeing since this time was characterized by the formation of follicles, 2 of the patients having been treated for trachoma. Usually one node is found between the lashes; at the same time conjunctivitis with many large follicles and with some papillary hypertrophy develops in the majority of cases. The removal of the nodes is quickly followed by full recovery from the conjunctivitis. Elschnig observed a corneal complication in the form of phlyctenular keratitis; Cavara [14] in the form of peripheral infiltration, and H. and S. R. Gifford, in the form of a corneal ulcer. The views on the pathogenesis of the conjunctival disease vary. H. and S. R. Gifford are, I dare say, right in asserting that the contents of the nodes of molluscum contagiosum coming in contact with the conjunctiva cause the ocular symptoms. This points to the infectivity of molluscum contagiosum. In fact, the condition is considered to be caused by the presence of a virus in an especially disposed organism. It cannot be due simply to the irritating effect of the desquamated epidermis. There is considerable difference between the conjunctival disease caused by molluscum contagiosum and that caused by the wart. Molluscum contagiosum usually produces a follicular, trachoma-like conjunctivitis, and involvement of the cornea is rare. The wart produces a simple subacute conjunctivitis or, often enough, keratitis of different types. The simplest explanation of this difference consists in the different biologic property of the viruses.

Cavara did not succeed in producing conjunctivitis with the pulp of the nodes of molluscum contagiosum dropped into the conjunctiva. I introduced a node into the lower fornix for three hours, and only a slight circumscribed hyperemia developed. To produce the disease, the conjunctiva must probably be infected with new virus masses. Bardelli [15] considered follicular conjunctivitis due to molluscum contagiosum as an allergic symptom, analogous to the conjunctivitis caused by atropine, physostigmine salicylate or chinondiimin (Ursolo), a hair dye. After a certain time, a period of sensitization during which these factors act, a conjunctival reaction suddenly manifests itself. The quick recovery after the agents had ceased to act also strengthens the view of Bardelli. A single infection of the eye with the virus of the other inclusion diseases of the skin—herpes, variola, variolovaccinia—may be sufficient to involve the cornea or conjunctiva. This can be proved by inoculating rabbit corneas with these viruses. Seven hours after inoculation with the herpes virus inclusions appear in the nuclei. Inoculation with the viruses of variola and variolovaccinia causes typical changes in the cornea in forty-eight hours. I examined conjunctival scraping obtained from subjects with conjunctivitis associated with wart. No inclusions were found in the epithelial cells.

14. Cavara, V.: Le congiuntiviti da mollusco contagioso, Boll. d'ocul. **3**:1, 1924.
15. Bardelli, L.: Congiuntiviti allergiche, Boll. d'ocul. **8**:817, 1929.

The period of incubation of the wart and molluscum contagiosum lasts for several weeks or months. But the virus cannot stay and multiply unchecked in the conjunctiva for so long a time. The conjunctiva is not covered with a protecting epidermis; the infected and loosened cells easily slough off; the circulation is active; the adenoid layer affords a protection against the penetrating virus and from here the virus entering the epithelium can be attacked. Thus the protecting forces of the conjunctiva are easily able to destroy a slowly multiplying virus with a long incubation period—easier and with less irritation than those of the other viruses with short incubation periods. But by renewing the attacks, the virus causes in the conjunctiva or cornea a permanent reaction, perhaps on an allergic basis, after the manner described by Bardelli in the conjunctivitis due to molluscum contagiosum.

This reaction varies according to the biologic properties of the virus. Merely the suddenly appearing and disappearing painful corneal symptoms, as in cases 1 and 6 and in the case of V. L., are suggestive of an allergic origin.

Removal of the growth stops the invasion of the virus, and consequently the irritation of the eye ceases.

SUMMARY

I have summarized my experiences with ocular diseases caused by common wart of the eyelid, described first by me in 1933 and by Vito in 1936. Seven cases of unilateral conjunctivitis, mostly of the subacute form, and 3 cases of keratitis of different types were observed in connection with warts of the eyelid. An additional case of bilateral conjunctivitis is reported in which warts occurred on the lids of each eye. The virus of the wart is considered as the etiologic factor in these cases. This view is based on the conformity of the following facts: The unilateral appearance; the involvement of the superficial layers of the eye, which points to an exogenous factor; the resistance toward the usual treatment; the presence of one or several warts on the margin of the lid of the diseased eye; the quick recovery after the removal of the warts, and the analogy to molluscum conjunctivitis. On the basis of these facts, I have come to the conclusion that all inclusion diseases of the skin are able to attack the eye.

OCULAR IMPORTANCE OF SARCOID

ITS RELATION TO UVEOPAROTID FEVER

FRANK B. WALSH, M.D.

BALTIMORE

Uveoparotid fever has received so much attention in ophthalmologic literature that the syndrome originally described by Heerfordt is diagnosed promptly. However, from time to time patients are encountered who lack one of the two signs considered essential to the diagnosis, namely, uveitis or parotitis, and in such cases the condition may remain undiagnosed. Another group of patients with a diagnosis of sarcoid may present, in addition to other signs underlying the diagnosis, either or both cardinal signs of uveoparotid fever. The relation between these two diseases has been considered in the recent literature by Longcope and Pierson [1] in their description of sarcoid; by Hamburger,[2] who was one of the first to recognize uveoparotid fever in this country; by Pautrier,[3] who expressed the belief that the two diseases are different manifestations of the same disease and are not due to tuberculosis, and by Bruins Slot, Goedbloed and Goslings,[4] who agreed with Pautrier that they are identical diseases but were of the opinion that both are due to tuberculosis.

Several case reports are summarized in this paper, but treatment is not considered. These studies have resulted in further clinical and his-

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

1. Longcope, W. T., and Pierson, J. W.: Boeck's Sarcoid (Sarcoidosis), Bull. Johns Hopkins Hosp. **60**:223-296 (April) 1937.

2. Hamburger, L. P.: Uveoparotitis, in Blumer, G.: The Practitioner's Library of Medicine and Surgery, New York, D. Appleton-Century Company, Inc., 1938, supp., chap. 12, pp. 118-124.

3. Pautrier, L. M.: Le syndrome de Heerfordt des ophtalmologistes n'est qu'une forme particulière de la maladie de Besnier-Boeck-Schaumann, Ann. de dermat. et syph. **9**:161-197 (March) 1938; Les lésions oculaires de la maladie de Besnier-Boeck-Schaumann, Arch. d'opht. **2**:689-696 (Aug.) 1938. Pautrier, L. M.; Jacob, and Weber: Maladie de Besnier-Boeck-Schaumann à forme uniquement pulmonaire et ganglionnaire sans manifestations cutanées, Bull. et mém. Soc. méd. d. hôp. de Paris **53**:1600-1608 (Dec. 20) 1937.

4. Bruins Slot, W. J.; Goedbloed, J., and Goslings, J.: Die Besnier-Boeck-(Schaumann-)sche Krankheit und die Uveo-Parotitis (Heerfordt), Acta med. Scandinav. **94**:74-79, 1938.

tologic evidence that uveoparotid fever and sarcoid are closely related diseases. The protean ocular manifestations of uveoparotid fever and sarcoid are exemplified in case reports.

CLINICAL-HISTOLOGIC CHARACTERISTICS OF SARCOID AND UVEOPAROTID FEVER

As an approach to the subject, it seems unnecessary to describe in detail the clinical characteristics of uveoparotid fever. However, it may be briefly stated that uveitis and parotitis are cardinal signs; transient palsies of the cranial nerves, usually involving the seventh nerve and always sparing the eleventh and twelfth nerves, may be observed; cutaneous lesions, often in the nature of erythema nodosum, are frequent; diabetes insipidus has been observed occasionally. The clinical characteristics of sarcoid are not so generally known. In English and American ophthalmologic literature scarcely any attention has been paid to sarcoid other than its occurrence in the eyelids, although Ernsting [5] in reporting such a case gave a comprehensive review of the literature. Ormsby [6] in his recent textbook defined sarcoid (multiple benign sarcoid; benign miliary lupoid-Boeck) as follows: "a disorder characterized by the formation of nodules and plaques, both cutaneous and subcutaneous, with frequent involvement of the bones (phalanges), glands and internal viscera, usually having a benign course and terminating after resolution of the lesions in atrophic, scar-like areas." Ormsby further stated that while clinical manifestations of sarcoid are usually seen in lesions of the skin, other organs and tissues may be affected; he mentioned involvement of nasal mucous membranes, tonsils, lymph nodes, bones, spleen, kidneys, lungs, liver and central nervous system, but, curiously enough, he failed to mention ocular involvement.

Sarcoid of the eyelids has been described frequently. In this situation sarcoid may or may not be associated with involvement of the adjacent tissues, such as conjunctiva, extraocular muscles or orbital tissue. Sarcoid of the eyelids has been described when other structures have been involved. Ernsting's patient presented coincident sarcoid of the lids and lesions on the legs which resembled erythema nodosum. When the lids or external structures of the eye are involved, spontaneous resolution often indicates the diagnosis, which, however, may be readily ascertained by biopsy of the tumor. The following summary of a case reported

5. Ernsting, H. C.: Boeck's Sarcoid of the Eyelid with Coexisting Darier-Roussy's Sarcoid: Report of a Case, with Review of the Literature, Arch. Ophth. **17**:493-504 (March) 1937.

6. Ormsby, O. S.: A Practical Treatise on Diseases of the Skin, Philadelphia, Lea & Febiger, 1937, p. 818.

by Wilmer[7] under the title "Tubercle-Like Nodules of the Episclera and Eyelids, Bilateral" illustrates involvement of the lids, conjunctiva and episcleral tissues with sarcoid.

CASE 1.—A woman, aged 56, complained of lumps in the eyelids. These were not painful to touch and were freely movable. There was a mass in the subconjunctival tissue over the right internal rectus muscle. Within two and a half months all the masses had disappeared with the exception of one in the lower lid of the right eye, which was removed surgically.

General physical examination gave negative results. Examination of the eyes revealed no abnormalities other than those mentioned. The vision was normal.

Histologic examination of the mass from the episcleral tissue showed it to be a small inflammatory nodule embedded in what appeared to be normal subconjunctival connective tissue. The nodule was sharply circumscribed and consisted for the most part of epithelioid cells with numerous giant cells and some sur-

Fig. 1 (case 1).—Nodules of the eyelids. (From Wilmer.[7])

rounding lymphocytic infiltration. No caseation was present. No bacilli were found in the growth. Treatment with tuberculin could not be credited with spontaneous resolution of the masses, which commenced while the dosage was infinitesimal.

The histologic picture of "hard tubercle" is characteristic of sarcoid. Evidence of caseation is unusual in cases of sarcoid, but it may be observed in figure 4, which is of special importance since it tends to support the idea that sarcoid is a manifestation of tuberculosis. The absence of tubercle bacilli in the lesions is characteristic of sarcoid. The patient was sensitive to 0.01 mg. of old tuberculin injected intradermally, although there is usually no reaction even to high concentrations of tuberculin.

7. Wilmer, W. H.: Tubercle-Like Nodules of Episclera and Eyelids, Bilateral, Tr. Am. Ophth. Soc. **31**:59-67, 1933.

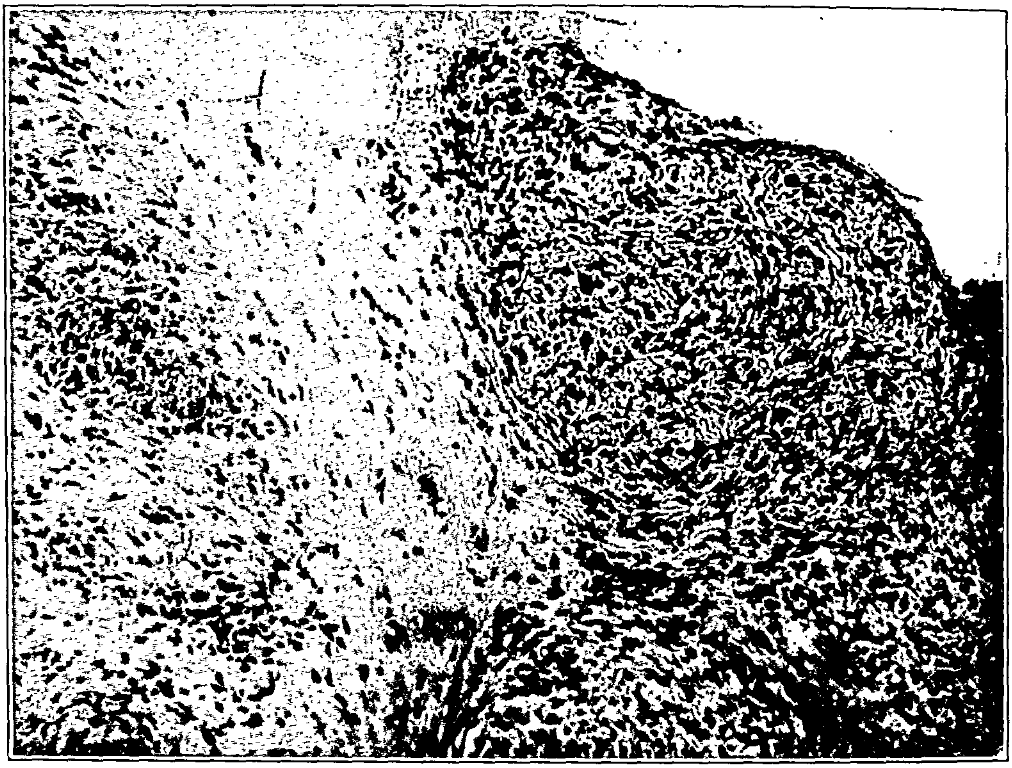

Fig. 2 (case 1).—Episcleral nodule. (From Tr. Am. Ophth. Soc. **31**:59, 1933.)

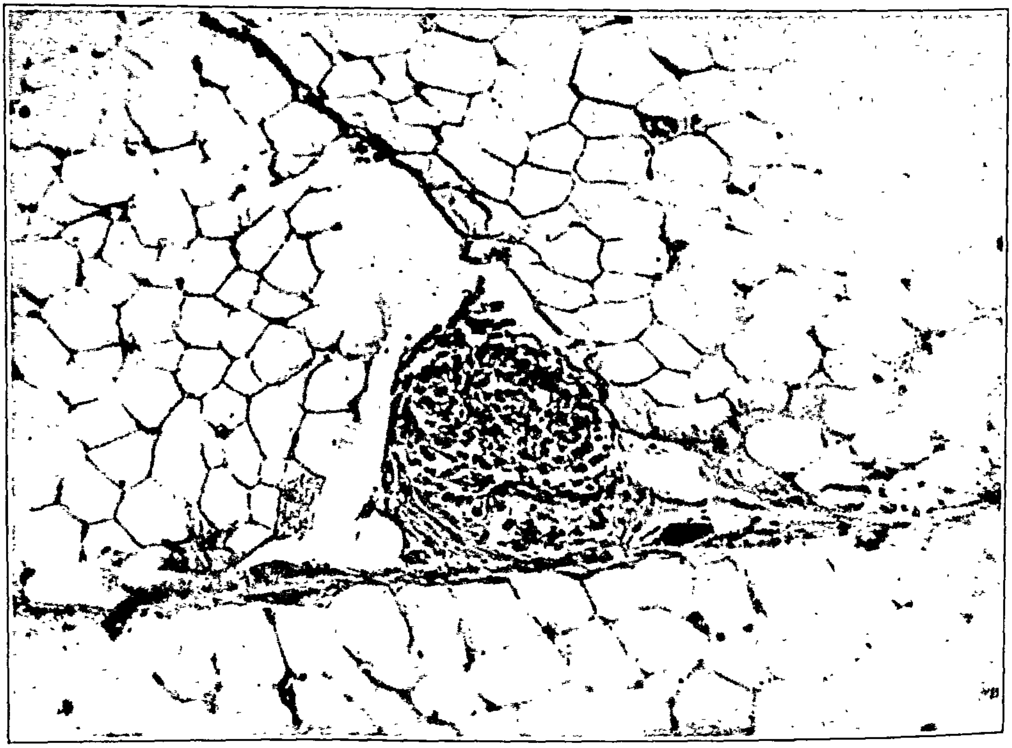

Fig. 3 (case 1).—Isolated tubercle in orbital fat. (From Tr. Am. Ophth. Soc. **31**:59, 1933.)

The next case illustrates bilateral swelling of the lacrimal glands as the only evidence of ocular involvement in widespread sarcoidosis affecting the skin and apparently responsible for enlargement of the liver and spleen.

CASE 2.—M. R., a Negress aged 38, was admitted to the Johns Hopkins Hospital on Aug. 29, 1938, complaining of a cutaneous rash of six months' duration and a lump in the abdomen of three years' duration. The family history was uninteresting. In 1926, when the patient visited a clinic because of a "sore throat," it was found that she had a positive Wassermann reaction of the blood. She failed to return for treatment and disappeared from observation.

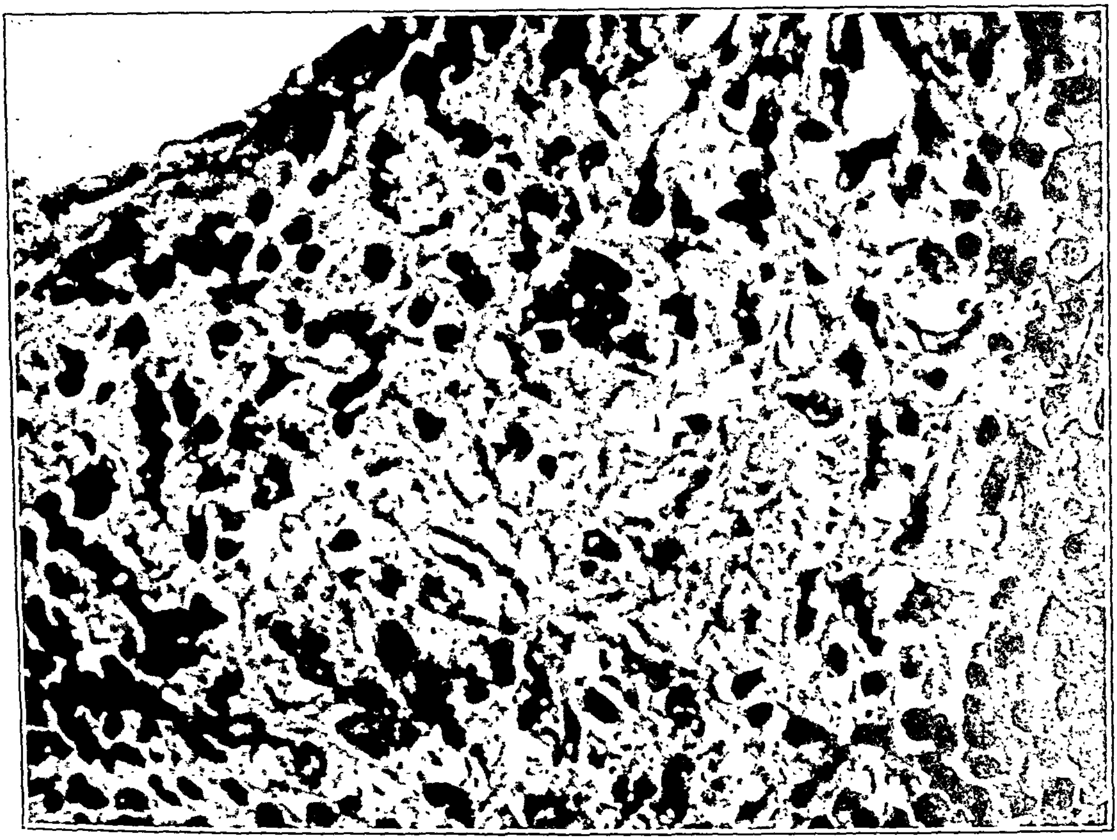

Fig. 4 (case 1).—Orbital nodule, showing beginning caseation; high power magnification. (From Wilmer.[7])

In 1935 she first noticed a mass in the left upper abdominal quadrant. The mass gradually increased in size but caused only slight discomfort. Early in 1938 a cutaneous eruption appeared over the abdomen and back and spread to the arms and legs. In April 1938 she was seen in a city dispensary, and because of the rash and a positive Wassermann reaction she was treated for secondary syphilis. Her condition did not improve, and in August she was transferred to the hospital. Examination showed the patient to be in no apparent distress. She had a low grade fever. Shotlike waxy brown papules were present on the arms. Over the trunk and thighs there was a maculopapular type of eruption made up of shotty papules, averaging about 0.5 cm. in diameter. The lesions did not cause discomfort. There was moderate enlargement of the superficial lymphatic glands, except the epitrochlear glands. The lacrimal glands were moderately enlarged. All the

swollen glands had a rubbery feel and were freely movable. There was pronounced enlargement of the spleen, which extended to 6 or 8 cm. below the costal margin. It was smooth and not tender. The liver was enlarged; its lower border was situated from one to two fingerbreadths below the costal margin. Examination of the eyes was negative except as already noted. Neurologic examination gave entirely negative results.

Roentgenograms of the lungs showed enlargement of the hilus nodes and infiltration at the base of the left lung and the apex of the right lung; infiltration of a lesser degree was present in the midzone and base of the right lung. Examination of the sputum was negative for tubercle bacilli. Roentgenograms of the bones of the hands and feet were normal. The Wassermann reaction on the blood was positive. There was no cutaneous reaction to 1 mg. of tuberculin. The Frei test was negative. The blood showed a moderate eosinophilia. There was a slight albuminuria. Section of a lymph gland and of the skin showed a characteristic picture of sarcoid.

The following case illustrates some of the characteristics and the usual clinical course of sarcoid. Except for an absence of involvement of the parotid gland, a diagnosis of uveoparotid fever might have been made.

Case 3.—L. H., a Negress aged 26, was observed during 1937. Her family history was unimportant. She had had mumps as a child. For five years she had frequent epistaxis and dyspnea on exertion. She had had three or four "needle" treatments while pregnant six years before her admission to the hospital.

During April 1937, both legs swelled up as far as the knees, and scalelike lesions developed on the thighs and forearms. She had a low grade fever. About June 1 inflammation of both eyes developed. On June 24 she was seen in the dispensary clinic, where a diagnosis of episcleritis was made. The vision was normal in both eyes. Slit lamp examination did not reveal any signs of intraocular inflammation. On June 28 a sudden hoarseness developed. The right vocal cord was paralyzed. Although the Wassermann reaction of the blood was negative, she was given antisyphilitic treatment. By July 22 the right vocal cord had regained its function, but the left vocal cord was paralyzed and the patient could not swallow because of paralysis of the soft palate. During the period from June, when she was hospitalized, until October she continued to have a low grade fever and frequent night sweats. The only abnormal physical finding, in addition to those mentioned, was slight enlargement of the liver. Early in October, by which time the paralysis had disappeared, she complained of a decrease in visual acuity and was found to have bilateral low grade uveitis, more severe in the left eye. Vision in the right eye was 20/20 and that in the left eye was 20/40. By the middle of October she had made a complete recovery, except that her vision remained slightly reduced in the left eye, and slit lamp examination continued to show minimal signs of inflammation in both eyes.

The Wassermann reaction of the blood and spinal fluid was negative on several tests. Roentgenograms of the chest showed marked enlargement of the mediastinal glands bilaterally, with a diffuse nontuberculous infiltration throughout both lungs. Biopsy of a section of skin showed tuberculosis cutis. Blood smears and blood counts were normal. The electrocardiogram was normal. Roentgenograms of the bones did not reveal any abnormality. The Frei test was negative.

Transient palsies of the nerves have been described frequently in cases of uveoparotid fever. The facial nerve is involved more frequently than any other. There are no cases reported of involvement of the eleventh or twelfth nerves. The facial paralysis is usually unilateral [8] but may be bilateral. The palsy is usually of the peripheral type, although central involvement has been observed; it seems probable that differentiation between central and peripheral involvement is not always attempted. Dr. Rowland H. Merrill,[9] of Salt Lake City, has had under his care a patient with a history similar to that in the foregoing case; with exacerbations of a chronic bilateral uveitis, transient palsies of the cranial nerves occurred during a period of several months. On one occasion the patient exhibited transient deafness.

The next case illustrates the occurrence of bilateral intraocular inflammation and tumors of the lid as well as general glandular enlargement. Biopsy of a gland showed sarcoid. The possibility of confusing the diagnosis of sarcoid with syphilis and the occurrence of outspoken pulmonary tuberculosis as a final lesion are particularly interesting.

Case 4.[10]—R. H., a Negro aged 28, had urethritis in 1928. In February 1934, after an attack of influenza, his eyes became inflamed. In March iridocyclitis was present; vision in the right eye was reduced to perception of hand movements and that in the left eye to 2/200. In April 1934 he was hospitalized with a diagnosis of bilateral syphilitic iridocyclitis with secondary glaucoma. The Wassermann reaction of the blood was positive. Antisyphilitic therapy was instituted on April 1, and by April 19 the visual acuity in the left eye had improved to 20/50 —1, but there had been no improvement in the vision of the right eye. The patient was given continued local treatment to the eyes and intense antisyphilitic therapy. During the course of treatment hard nodules appeared in his lids. The inflammatory condition of his eyes gradually improved, and after several weeks he was discharged from the hospital with the masses in his lids unchanged. He visited the ophthalmologic and syphilis clinics regularly thereafter.

In February 1936 there was no perception of light in the right eye and vision in the left eye was 20/30. In May 1936 the patient was again hospitalized because of signs of consolidation in his right lung. At that time he had a low grade fever. Masses in the eyelids and general glandular enlargement were still present. The nature of the pulmonary trouble was not clear. Roentgenograms suggested an abscess of the lung. There was a diffuse infiltration and partial consolidation of the midportion of the right lung, which was associated with considerable pleural reaction. There was no cavitation. The sputum was constantly negative for tubercle bacilli.

Special tests were made. Biopsy of a gland from the groin and of one of the masses on the lid showed lesions which were typical of sarcoid. The patient gave a positive reaction only to 1 mg. of old tuberculin.

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8. Evensen, O. K.: Uveoparotid Fever, Nord. med. tidskr. **14**:2019-2023 (Dec. 11) 1937.

9. Merrill, R. H.: Personal communication to the author.

10. This case has been previously reported by Longcope and Pierson [1] as case 8.

Fig. 5 (case 4).—Appearance of a hard tubercle; low power magnification.

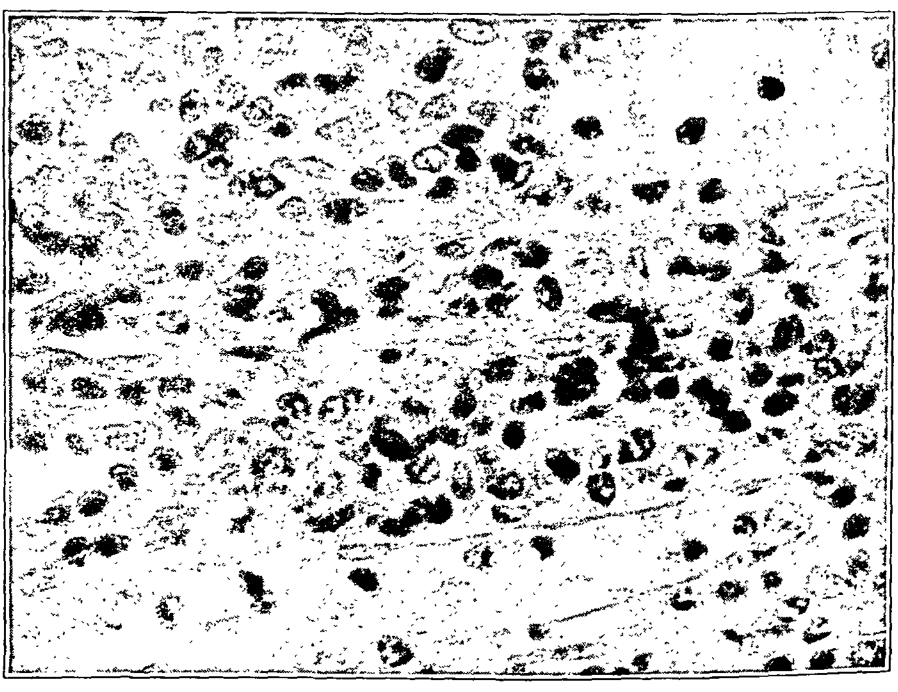

Fig. 6 (case 4).—Section showing epithelioid cells and lymphocytes; high power magnification.

After several weeks the symptoms of pulmonary sepsis disappeared spontaneously, although physical signs of pulmonary involvement persisted. The general glandular enlargement and masses in the eyelids were unchanged up to the time the patient was discharged from the hospital.

In March 1937 the patient had a temperature of 100 F.; his pulse rate was 120. He had been treated with general irradiation with ultraviolet rays for over a year. He complained of weakness, cough and loss of weight. The glands, which had previously been swollen, had decreased in size. Examination of the chest suggested advanced tuberculosis, and tubercle bacilli were found in the sputum. Roentgenograms revealed soft fibroid infiltration of the right lung, with cavitation in the midportion.

Although lesions typical of sarcoid rarely if ever contain tubercle bacilli, it is not uncommon that definitely proved tuberculous lesions may be coincidental or terminal. Garland and Thomson [11] and Souter [12] reported cases in which patients suffering from uveoparotid fever died of miliary tuberculosis. Wiseman [13] reported a case in which sections of glands showed typical sarcoid, and scrapings from ulcers on the leg contained tubercle bacilli. In Hamburger and Schaffer's patient with uveoparotid fever genitourinary tuberculosis developed thirteen years after apparent recovery.[14]

The description of the roentgenographic picture of the thorax in cases of sarcoid and of uveoparotid fever has been similar. The essential changes are enlargement of the lymph glands at the hilus and scattered parenchymal lesions or both. Frequently the differential diagnosis must include miliary tuberculosis because of the appearance of the film. In both uveoparotid fever and sarcoid the outstanding feature is the definite involvement of pulmonary tissue and the absence or slightness of the resultant symptoms. In both diseases verified miliary tuberculosis has been recorded as a terminal stage, although in the majority of such cases the patients recover completely.

Another case illustrates confusion in diagnosis between Hodgkin's disease, syphilis and sarcoid. In this case recognition of osseous changes characteristic of sarcoid indicated the proper diagnosis.

CASE 5.[15]—T. M., a Negro boy aged 14 years, in 1925 had bilateral painless swellings of the parotid glands and was thought to have mumps. The glands continued to enlarge slowly on both sides of the neck. In February 1926 he was

11. Garland, H. G., and Thomson, J. G.: Uveo-Parotid Tuberculosis, Lancet **2**:743-746 (Oct. 6) 1934.

12. Souter, W. C.: Diseases of the Choroid: A Case of Uveo-Parotid Fever with Autopsy Findings, Tr. Ophth. Soc. U. Kingdom **49**:113-127, 1929.

13. Wiseman, R. H.: Multiple Benign Sarcoid and Tuberculous Ulceration, Brit. M. J. **1**:673-674 (March 26) 1938.

14. Hamburger, L. P., and Schaffer, A. J.: Uveoparotid Fever as a Manifestation of Mikulicz's Syndrome, Am. J. Dis. Child. **36**:434-444 (Sept.) 1928.

15. This has been previously reported by Longcope and Pierson [1] as case 2.

hospitalized. He was afebrile. Roentgen examination showed enlargement of the mediastinal glands. The lacrimal glands were enlarged. On March 3, 1926, the left eyelids became swollen, and the swelling extended to the bridge of the nose. There was an accompanying mild febrile reaction. The swelling of the glands cleared rapidly. Biopsy of tissue from the cervical and axillary glands showed tubercles without caseation. Tubercle bacilli were not found in the tissue. The patient was discharged from the hospital without any pronounced improvement. Treatment with ultraviolet rays was given three times weekly. In the summer of 1926 his condition improved rapidly.

In 1930, when he was 18 years old, he was admitted to the hospital complaining of an eruption of six months' duration on his back, the sides of his neck, his face and his eyelids. The cutaneous lesions were a deep pink, varying from round to oval, and were seen in some instances to be discrete and in others to be confluent. Some of the lesions on the neck, face, eyelids and pubic region were 0.5 cm. in length. Nodules were present on the hard palate, where they were yellow and raised. The cervical glands at the angles of both jaws were enlarged to the size of a pigeon's egg. There was some general glandular enlargement. The patient was given antisyphilitic treatment because of a positive Wassermann reaction, but no change occurred in the cutaneous lesions. Clinical and laboratory examinations gave essentially negative results. A biopsy caused the cutaneous lesions to be classified as tuberculids. A roentgenogram of the chest showed consolidation in the apex of the right lung.

In 1936 the Wassermann reaction of the blood was negative. The patient was then 23 years old. Scars of the previously described cutaneous lesions were apparent over both arms and were from 1 to 4 cm. in diameter. There was a keloid-like lesion on the back of the neck, 10 cm. long and 4 cm. wide, which was elevated and purplish, and on its surface were small, yellowish white pinhead points. Three similar but smaller lesions were present on the back. These were 2 by 3 cm. in size. The hands showed dorsal depressions in the middle phalanges, and on the first and third fingers and the thumb of the left hand the terminal phalanges appeared eroded and healed, leaving small nails. All the fingers were hyperextensible. Roentgenograms of the hands showed multiple lesions of the fingers involving the diaphysis; some of the joints showed areas of destruction, absorption and adjacent sclerosis associated with little soft tissue reaction. The patient was treated with ultraviolet rays.

On April 6, 1937, the patient was again seen. He had had inflammation of the left eye for the past year. For nine months he had had impaired hearing on the left. He complained of a large scar on the back of the neck (keloid), which he wished to have removed. Examination of the ear showed bulging of the ear drum with deafness of the middle ear type; this was probably due to the presence of sarcoid tissue within the ear.

The patient was tested only with low dilutions of tuberculin, to which he did not show a reaction.

The bony changes from sarcoid are shown in figure 7. Connolly [16] issued a warning that an entirely normal roentgenographic picture does not eliminate the possibility of sarcoid tissue being present in the bones, as biopsies of bony tissue have revealed such changes when the roent-

16. Connolly, A. E.: Osteitis Tuberculosa Multiplex Cystoides and Sarcoid Lesions, Brit. J. Radiol. **11**:25-37 (Jan.) 1938.

genograms were apparently normal. He described the changes characteristic of sarcoid as being either diffuse or circumscribed. Recent lesions may be characterized by a slightly diffuse enlargement of the bone and by lack of differentiation between the medulla and the compact substance, which may be riddled with small rounded cavities or reduced to paper thinness; cystlike areas may be present in the head of the bone; solitary cystlike cavities are generally of large size and suggest the confluence of smaller ones; they may be central or peripheral in position

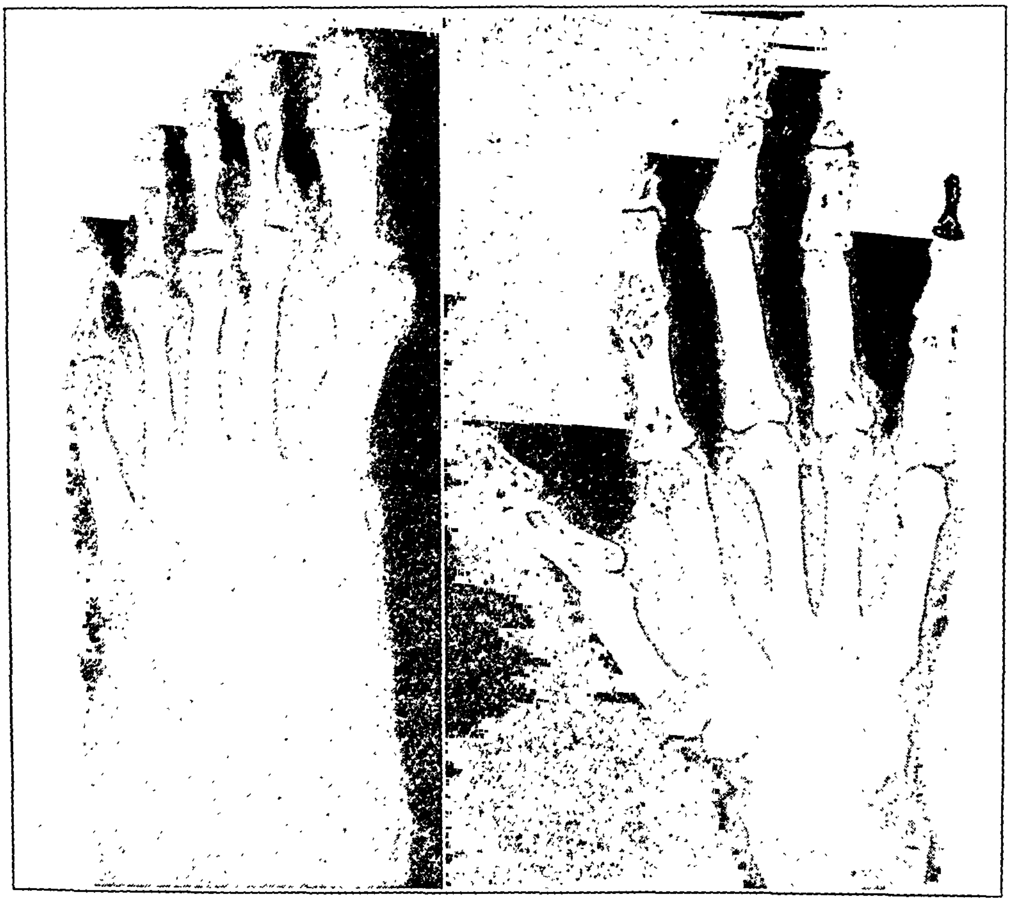

Fig. 7.—Roentgenographic changes in a case of sarcoid. (From Longcope and Pierson.[1])

(Connolly). The long bones of the hands and feet are frequently involved.

SARCOID INVOLVING THE CENTRAL NERVOUS SYSTEM

The cause of transient palsies of the cranial nerves in uveoparotid fever is not clear. The facial nerve is most frequently affected, and the paralysis is usually of the peripheral type. However, involvement of brain tissue and meninges has been observed in cases reported as cases of sarcoid and uveoparotid fever.

In the next case the diagnosis of sarcoid rests mainly on the clinical history but, in addition, is supported by a microscopic examination of tissue from an enucleated eye. The case is reported in greater detail than the preceding ones, because it demonstrates involvement of the uveal tract and the central nervous system. In this patient bilateral atrophy of the optic nerve was observed ten years before it became necessary to enucleate the eye from which the specimen was obtained. In the remaining eye there was an unusual ophthalmoscopic appearance, and it seems possible that such a picture may be characteristic of intraocular sarcoid.

Case 6.—M. F., a white woman aged 34, was examined by me in April 1938. She complained of loss of vision in the left eye for several years and of recent decrease in vision in the right eye. Her family history was noncontributory. She was entirely well until she was 16 years of age. She first menstruated at 14 and continued to do so at regular intervals until she was 16, when menstruation ceased spontaneously. At 20, she married. Her health remained good until she was 22 years old. Menstruation did not occur for several years and then only at long intervals. For three years, however, she had been taking various glandular extracts and had menstruated fairly regularly.

In 1926 she complained of redness of the left eye, which persisted. She consulted an ophthalmologist, who made a diagnosis of uveitis but also observed atrophy of the optic nerve in that eye. In 1927 she consulted Dr. Alan C. Woods, who found in the right eye vision of 20/30 —, blurring of the temporal margin of the nerve head with slight temporal pallor, numerous superficial discrete white spots in the paramacular area and normal vessels; in the left eye there was a severe inflammation, as evidenced by congestion of the conjunctival vessels, irregular bound down pupil and hazy media; there was pronounced atrophy of the optic nerve, and white spots scattered throughout the fundus were seen with difficulty. The visual field of the right eye showed a definite constriction of the upper temporal quadrant, and that of the left eye consisted only of a small nasal island. Dr. Woods believed that the condition was probably ocular tuberculosis but stated the atrophy of the optic nerve and changes in the visual field suggested the possibility of a tumor of the pituitary gland. Roentgenograms of the sella turcica were normal. In 1928 cutaneous lesions developed, discrete masses being present in the skin over the nose and on the arms, the face and the right buttock. A dermatologist diagnosed the lesions as tuberculids. These cleared in from two to three years, during which time various forms of treatment were used. In 1929 the patient saw Dr. Wilmer, who agreed that she had tuberculosis of the left eye; ophthalmoscopic examination of that eye revealed a mass in the choroid, which was elevated about 8 diopters; there was some pallor of the temporal side of the right nerve head. After this examination the patient felt that the left eye was hopelessly lost and did not seek further advice until 1938, when she thought she required new glasses. She was told she had pronounced atrophy of the optic nerve in the right eye and that the visual field was contracted.

She was then admitted to the Johns Hopkins Hospital. Stereoscopic roentgenograms of the skull were normal, and the optic foramens were of normal size. Roentgenograms of the bones of the skeleton and of the lungs were normal. General examination showed that the patient had diabetes insipidus, which, however,

Fig. 8 (case 6).—Appearance of patient's eye in 1929.

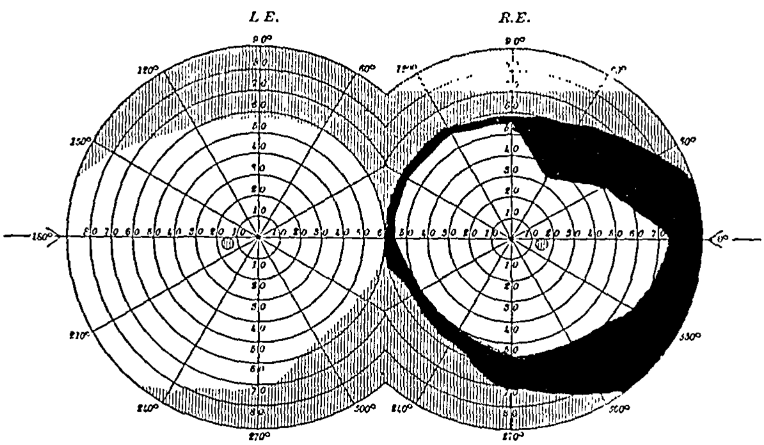

Fig. 9 (case 6).—Visual fields of patient taken on Nov. 27, 1927, with a 1 degree white test object. Vision in the right eye was 20/30 —.

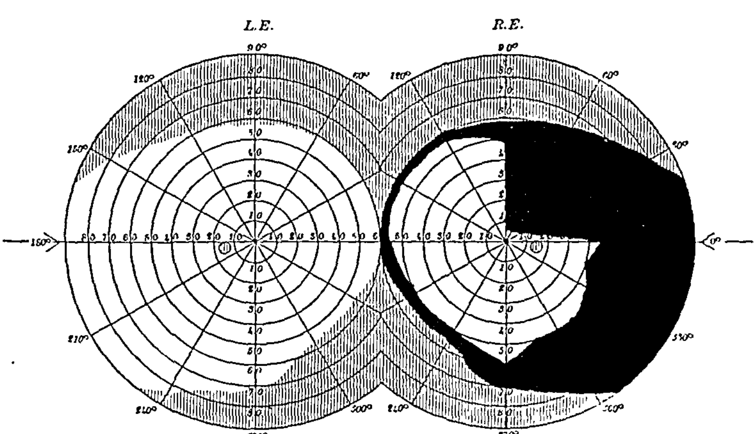

Fig. 10 (case 6).—Visual field of the patient taken on Sept. 13, 1938, almost eleven years later, with 1 and 2 degree test objects, showing the increase in the defect. Vision in the right eye was 20/20—2. The visual acuity has remained normal in spite of considerable atrophy of the optic nerve.

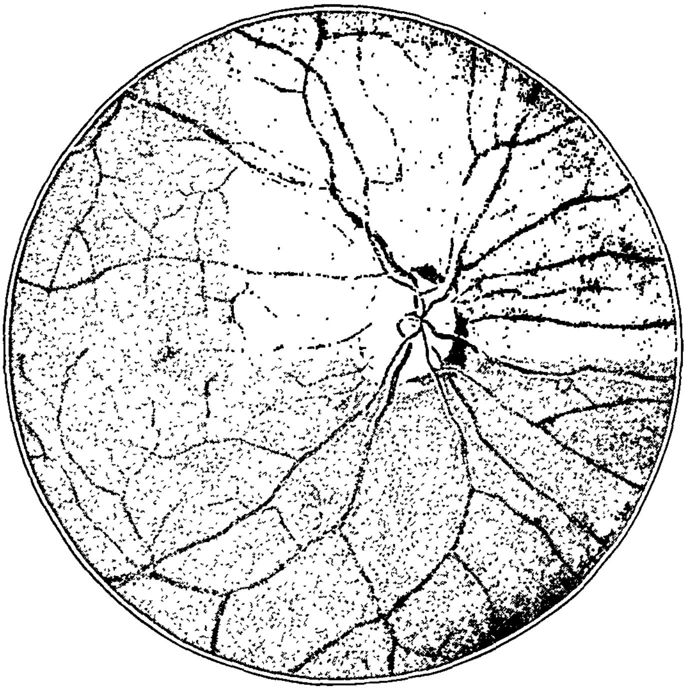

Fig. 11 (case 6).—Photograph of a painting of the fundus of the right eye. Note the tendency to a perivascular situation of the whitish yellow masses.

Fig. 12 (case 6).—Typical sarcoid tumor obtained in serial section of the enucleated left eye.

was readily controlled by the administration of pituitary extract. Laboratory examinations, including the Wassermann reaction of the blood, gave entirely negative results. The patient had a slight positive reaction to 0.01 mg. of old tuberculin.

Examination of the eyes showed the right eye to be normal externally. The left eye was small and phthisic, with grayish opacities extending into the cornea from the adjacent sclera. The lens was cataractous. Vision in the right eye was 20/60; with a + 3.25 sph. $\supset$ + 0.75 cyl., axis 180° vision was 20/25 + 3, and the patient could read Jaeger test type 1 at 10 inches (25.5 cm.). There was no perception of light in the left eye. The visual fields are shown in figures 9 and 10. The ophthalmoscopic appearance of the right fundus is shown in figure 11. Slit lamp examination of the right eye gave negative results. In the left eye there were opacities and vascularization deep in the cornea, an atrophic irregular iris and a cataractous lens.

It was thought that sarcoid was the proper diagnosis. The sequence of events was instructive. The occurrence of unexplained amenorrhea was followed by intraocular inflammation and coincident atrophy of the optic nerve; cutaneous lesions (unfortunately the report of a biopsy obtained elsewhere has been lost) which were diagnosed as tuberculids had disappeared, and only scar tissue remained as evidence that they had existed; progress of the atrophy of the optic nerve in the remaining eye occurred with an increase in the defect in the visual field; diabetes insipidus was discovered; sarcoid was demonstrated in the enucleated eye.

Reis and Rothfeld [17] reported a similar case in a 17 year old girl who had suffered from sarcoid and in whom bilateral papilledema developed which was followed by atrophy of the optic nerve in one eye; in the other eye a mass was observed to lie in front of the disk, and smaller masses were observed in the optic nerve itself; autopsy showed infiltrating masses involving the optic nerves, chiasma, cerebral peduncles and left temporal lobe; histologic examination showed the masses to be made up exclusively of epithelioid cells. Autopsy was recently performed in a somewhat similar case at the Johns Hopkins Hospital and is being reported by Dr. William Vandergrift. Sarcoid masses were present in the brain and meninges, and there was a bilateral papilledema.

RÉSUMÉ

The clinical and histologic characteristics of sarcoid have been exemplified in the preceding case reports, and it is to be noted that although ocular tissues were involved in all the cited cases, many cases of sarcoid occur in which the eyes are not affected. It seems unnecessary further to labor the point that uveoparotid fever and sarcoid are similar diseases and may be different manifestations of the same disease.

17. Reis, W., and Rothfeld, J.: Tuberkulide des Sehnerven als Komplikation von Hautsarkoiden vom Typus Darier-Roussy, Arch. f. Ophth. **126**:357-366, 1931.

Although uveoparotid fever has not been discussed as such and many comprehensive bibliographies on the subject are readily available, a brief selected group of references are included for the reader.[18]

THEORIES AS TO CAUSE OF SARCOID AND UVEOPAROTID FEVER

Main interest centers around the question as to whether or not sarcoid and uveoparotid fever are due to tuberculosis. The weight of opinion undoubtedly favors the view that tuberculosis causes the conditions, but this has not been proved beyond question. They may be due to attenuated organisms. Certainly in reported cases of both uveoparotid fever and sarcoid the histologic appearance of the characteristic lesion—masses of epithelioid cells with occasional giant cells, sometimes surrounded by a ring of lymphocytes and without caseation, a histologic picture of "hard" tubercle—has been reported with almost monotonous regularity. The designation of paratuberculosis by Weve [19] for such cases may have some value in referring to the similarity of these lesions to the usual lesions of tuberculosis in which tubercle bacilli can be demonstrated. Case 1 here reported is of interest in that one section revealed evidence of caseation. Case 3 is even more noteworthy in that the patient who had the characteristic lesions of sarcoid, as shown both by biopsy and roentgenographically, later had frank pulmonary tuberculosis. Even more striking is the occurrence of genitourinary tuberculosis in Hamburger and Schaffer's patient after an apparent freedom from symptoms over a period of thirteen years. Kyrle [20] discovered acid-fast bacilli in early lesions, but the bacilli disappeared when the sarcoid became developed. Garland and Thomson, who introduced the term uveoparotid tuberculosis, and Souter described cases in which death was ascribed to miliary tuberculosis. Pinner [21] included both uveo-

18. Critchley, M., and Phillips, P.: Uveo-Parotitic Paralysis and Iridocyclitis, Lancet **2**:906-907 (Nov. 1) 1924; cited by Folger, H. P.: Uveoparotitis (Heerfordt): Report of a Case, Arch. Ophth. **15**:1098-1116 (June) 1936. Feiling, A., and Viner, G.: Iridocyclitis-Parotitis-Polyneuritis, J. Neurol. & Psychopath. **2**: 353-358 (Feb.) 1922. Heerfordt, C. F.: Ueber eine "Febris uveo-parotidea subchronica" an der Glandula parotis und der Uvea des Auges lokalisiert und häufig mit Paresen cerebrospinaler Nerven kompliziert, Arch. f. Ophth. **70**:254-273, 1909. Levin, P. M.: The Neurological Aspects of Uveo-Parotid Fever, J. Nerv. & Ment. Dis. **81**:176-191 (Feb.) 1935. Savin, L. H.: An Analysis of the Signs and Symptoms of Sixty-Six Published Cases of the Uveoparotid Syndrome, with Details of an Additional Case, Tr. Ophth. Soc. U. Kingdom **54**: 549-566, 1934. Tait, C. V. B.: Uveo-Parotitis, Lancet **2**:748-749 (Oct. 6) 1934.

19. Weve, H.: Familiäre pseudotuberkulöse symmetrische Entzündung der Speichel- und Tränendrüsen und der Uvea (Mikulicz-Heerfordt), Ztschr. f. Augenh. **60**:68, 1926.

20. Kyrle, J., cited by Pinner.[21]

21. Pinner, M.: Noncaseating Tuberculosis: Analysis of the Literature, Am. Rev. Tuberc. **37**:690-728 (June) 1938.

parotid fever and sarcoid as examples of noncaseating tuberculosis; he collected reports of 17 autopsies in cases of sarcoid and added 1 of his own; in 4 cases pulmonary tuberculosis was the cause of death, and in a few others less significant tuberculous foci were found in addition to the main lesions. Some arguments in favor of a tuberculous etiology are: (*1*) the occurrence of tuberculous lesions elsewhere in the body; (*2*) the histologic character of the lesions except for the absence of caseation, even this being observed occasionally; (*3*) the transformation into classic tuberculosis, as observed in case 4, and (*4*) the disappearance or alleviation of sarcoid lesions with the development of frank tuberculosis, as observed in case 4 in which the glandular swelling decreased coincident with the onset of outspoken tuberculosis. Pinner expressed the belief that the "hard tubercle" should be considered a phase of tuberculosis rather than a type of tuberculosis and that tubercle bacilli may be found in the very early lesions. His article brings this subject up to date.

Absence of sensitivity to tuberculin or reduction of sensitivity is observed frequently in cases of sarcoid. In the absence of definite knowledge regarding the etiology of sarcoid, the explanation of this constant reduction in sensitivity is not clear. If, however, sarcoid is an atypical form of tuberculosis, as it well may be, the lack of sensitivity to tuberculin and the usual absence of caseation are what might be expected. This statement is based on Rich and McCordick's work, as outlined in the chapter on tuberculosis in Woods' book on allergy and immunity.[22] Rich and McCordick give the following formula: $\text{Lesion} = \frac{\text{Virulence} \times \text{Number} \times \text{Degree of Allergy}}{\text{Resistance}}$.

If in this formula it is assumed that the organisms are few in number and are of low virulence and that there is a slight degree of allergy and a high degree of resistance, the histologic and clinical features of sarcoid are what might be expected. The occurrence of caseation as exemplified in case 1 (fig. 4) possibly accounts for the increased sensitivity to tuberculin.

Another point of extreme interest is the possible relation of syphilis to uveoparotid fever and sarcoid. It will be noted that in 3 of the 6 cases here reported there were positive changes in the blood. In several cases of uveoparotid fever reported from this hospital by Thompson[23] the patients had positive Wassermann reactions, but as he was working in the syphilis department it was thought that coincident syphilis was not remarkable. The possibility of noncaseating tuberculosis resulting in positive but nonspecific changes in the blood has been raised by Gold-

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22. Woods, A. C.: Allergy and Immunity in Ophthalmology, Baltimore, Johns Hopkins Press, 1933, pp. 98-133.

23. Thompson, W. C.: Uveoparotitis, Arch. Int. Med. **59**:646-659 (April) 1937.

berg and Cohen,[24] who reported a case of sarcoid. Stokes [25] is authority that even the refined technic of the modern Wassermann test does not eliminate this possibility. Dr. J. Earle Moore [26] of the syphilis department of the Johns Hopkins Hospital agreed that such false positive reactions may occur; he further remarked that an investigation on this subject is being made at the present and that the report of the commission undertaking the investigation may prove interesting on just this point. Syphilis has been suggested as a cause in a minority of cases by Nicolas and Gaté.[27]

CONCLUSIONS

1. Sarcoid and uveoparotid fever are closely allied conditions for which the cause is not known, but it will probably be proved that they are due to tuberculosis.

2. The symptom complex is of particular interest to the ophthalmologist because of the frequent involvement of ocular tissue which may be manifested at any time during the course of the disease.

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24. Goldberg, L. C., and Cohen, S.: Darier-Roussy Sarcoid: Report of a Case, J. Med. **19**:73-75 (April) 1938.

25. Stokes, J. H.: Modern Clinical Syphilis, ed. 2, Philadelphia, W. B. Saunders Company, 1934, p. 131.

26. Moore, J. E.: Personal communication to the author.

27. Nicolas, J., and Gaté, J.: A propos des sarcoïdes, Bull. Soc. franç. de dermat. et syph. (Réunion dermat., Strasbourg) **41**:1011-1017 (June) 1934.

SPATIAL DISORIENTATION WITH HOMONYMOUS DEFECTS OF THE VISUAL FIELD

MARK KANZER, M.D.

AND

MORRIS B. BENDER, M.D.

NEW YORK

Patients with homonymous hemianopia differ in the degree to which they are aware of their defects. When the lesion is infrageniculate, e. g., in the optic tract, the existence of partial blindness is recognized, and a compensatory turning of the head and eyes develops. If an adjusted attitude is not established, the patient stumbles against objects on the blind side and overlooks words and letters in reading. The reading difficulties are less likely to be apparent in longer sentences because the resulting change in meaning arouses the attention of the patient and he adjusts his gaze until the proper field of vision is achieved. In some cases of lesions of the suprageniculate pathways the visual disturbances may be complicated by agnostic factors. The patient shows faulty comprehension of visual objects and consequently is unable to compensate automatically for his defects.

Kleist distinguished between visual "object agnosia," in which the forms of objects are not recognized, and "spatial agnosia," in which forms are correctly perceived but difficulty in spatial orientation occurs. Spatial agnosia usually is associated with homonymous hemianopia but may be found in the presence of normal visual fields. Studies by Bálint,[1] Fuchs [2] and other observers have emphasized the abnormal distribution of attention which occurs in this condition, manifesting itself in such disorders as dyslexia, simultaneous agnosia and constructive apraxia. The visual and psychologic aspects of this form of agnosia are the subject of our investigation.

REPORT OF A CASE

Spatial disorientation was found in a patient with defects in the left homonymous field of vision and symptoms of a tumor in the right cerebral hemisphere. An operation revealed an infiltrating spongioblastoma multiforme in the right temporal lobe. The extent of the tumor could not be determined.

From the Neurological Service of Dr. Israel Strauss, Mount Sinai Hospital.

1. Bálint, R.: Seelenlähmung des Schauens, optische Ataxie, räumliche Störung der Aufmerksamkeit, Monatschr. f. Psychiat. u. Neurol. **25**:51, 1909.

2. Fuchs, W.: Untersuchungen über das Sehen der Hemianopiker und Hemiamblyopiker, Ztschr. f. Psychol. **84**:67, 1920.

The spatial orientation of the patient was investigated as soon as he was sufficiently recovered from the operation. The charted visual fields at this time showed an almost complete homonymous hemianopia with partial retention of vision in the left inferior quadrants (fig. 1). There was a macula-splitting hemichromatopia. The fundi showed papilledema of 3 diopters bilaterally. The spontaneous behavior of the patient showed a disposition to seek the right side and to ignore the left. He would direct his attention only to the right columns of a newspaper. In describing pictures, he would select details on the right. In writing, he would leave the left half of the page blank (fig. 2). He could read letters and short words correctly, but longer words caused difficulty, and his eyes could not follow a printed line. With a series of letters he had a tendency to omit one or more letters on the extreme left. Thus DC was read as C, WDC as DC and FEAR as EAR. When the patient was first seen it was thought that he could not count fingers at a distance of a few feet. Careful investigation revealed that he simply ignored the finger at the extreme left. Examination with a Snellen chart showed a visual acuity of 8/20 (uncorrected) in each eye.

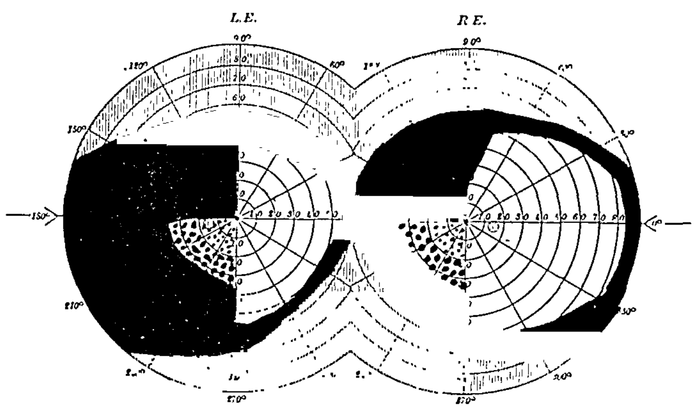

Fig. 1.—Postoperative visual fields. The small dots represent the areas in which a 2 mm. white object was recognized; the large dots represent areas in which 10 mm. white objects were still recognized. The interrupted lines indicate a left hemichromatopia.

Analysis of the patient's difficulties in perception revealed the influence of the configuration with respect to the fixation point. There was a tendency visually to bisect symmetric figures, so that the right half was perceived while the left half was ignored. If the equilibrium of the figure was to the left, the entire figure was seen; if it was to the right, details on the left were not observed. Simple geometric figures and familiar forms in which details on the left were intentionally obliterated by the examiners were perceived by the patient as complete. These tendencies were illustrated in the tests to be described.

An incomplete M was shown (fig. 3 *a*), and the patient was asked to fill any gaps he saw. He promptly filled the space on the right but ignored that on the left. Next the figure of a 4 was presented with a gap (fig. 3 *b*), which the patient was to fill. He completely ignored the detached figure to the left and constructed a 4 entirely from the figure on the right (fig. 3 *c*). He was instructed to convert an F into an E. This was done, but instead of merely adding a line to the F, he retraced the entire figure and then added the line that made it an E.

When shown a primitive sketch of a face (*fig. 3 d*), the patient promptly recognized what it portrayed. When shown similar sketches in which features on the right were omitted (an eye and an ear), he perceived the absence of these features. However, when features on the left were omitted, he was not aware of their absence. He was asked to point to the left eye in one figure in which it was absent. He took a pencil and pointed somewhat to the left of where the eye belonged and insisted that this feature was present.

He also showed inability to halve a line properly, the left half of the line always being longer than the right. When he could be persuaded to fix his eye

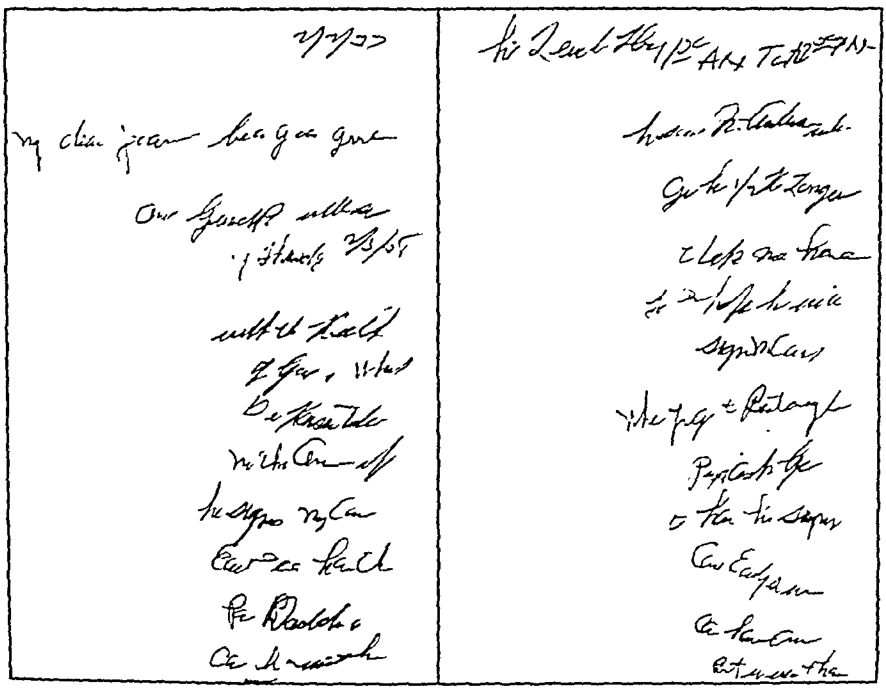

Fig. 2.—A letter written by the patient. The left half of the page remains blank.

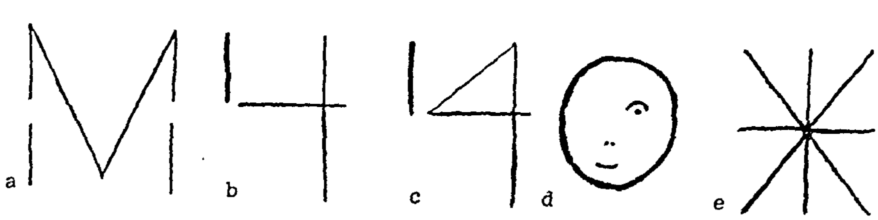

Fig. 3.—Tests given to patient. With *a*, the gap on the right was recognized and filled; the gap on the left was overlooked. An incomplete 4, as in *b*, was next shown to the patient. The 4 was completed by the patient as indicated by *c*. The part of the figure to the left was ignored. With *d*, a sketch of a face, the patient claimed to see both eyes. The figure indicated by *e* was described by patient as a K.

on a point, the letters to the right were always read correctly, and the patient stated that the letters farther to the right were clearer than those nearer the fixation point, a finding similar to that of Fuchs in an analogous case for which he postulated the existence of a "pseudofovea." It could also be shown that when the patient continued to fix a point there was no omission of letters or parts of

figures to the right of the point. Lines were also halved correctly when the patient fixed his attention to the left of them.

Of great interest was the way in which pictures were perceived. The patient was shown a number of photographs. Each time his attention turned spontaneously to the extreme right. When shown the picture of an automobile with figures in it, he began to describe what he saw. "That is a radiator; there is a wheel; that is a very fine car." (Do you see anything else in the picture?) "There is a tree; there is a door" *(indicating objects at the right)*; "the car is in front of the door. I guess some one wants to get out." (Is there anything else that you see?) "Oh, there is a woman in the car; the man is talking to her. I guess they are going out together."

This type of reaction is an excellent illustration of "optic inattentiveness" in which the person, usually with homonymous hemianopia, spontaneously ignores objects toward the defective field but becomes aware of them when he is specifically reminded of their existence. In this case the patient not only tended to concentrate his attention to the right; he spontaneously halted his description at the middle of the picture. His answers also revealed other characteristics of great interest. He would seize on isolated details of the picture—the radiator, the wheel, a tree. This tendency is of the type described by Wolpert as "simultaneous agnosia." He would also readily spin a story about the details which he grasped.

He was able to copy simple designs but overlooked parts of the figure to the left; in copying a Y, he omitted the left branch; in copying a square, he overlooked the left side. When shown a star tachistoscopically (fig. 3 *c*), he reported that he saw a K. Although he told time readily, he was incorrect, and it was obvious that he did not actually see the position of the hand on the left side of the dial. Tests showed no disturbances in arithmetical reasoning. Attempts to construct figures resulted in errors such as were described by Kleist in his cases of "constructive apraxia."

It was observed during repeated examinations that the performances improved. The patient became aware of many of his difficulties and learned to overcome them by compensation. He would no longer read spontaneously but would carefully study the pages and keep turning his head until the entire word or line came within his view. Other sensory spheres were utilized to compensate for the defective vision, as in the recognition of people by listening to their voices.

Mental examination disclosed good orientation in all spheres. Special tests showed a slight impairment of recent memory. The patient was cooperative, but there was emotional instability, as manifested by frequent spells of weeping. There was little insight as to the serious nature of his illness, and the visual disturbances seemed to cause the patient little inconvenience or concern.

COMMENT

Studies on "spatial agnosia" repeatedly confirm the significance of disturbances in attention. The early case of Bálint [1] provides an interesting illustration. The patient, despite an intact visual field, disregarded all objects which were not at an angle of 35 to 40 degrees to the right. He could perceive only a single object at a time and noticed others almost adjacent only when his attention was specifically called to them. The size of the objects was of little significance.

The deviation and the general constriction of attention appear to be characteristic features of spatial disorientation. A valuable study by Fuchs [2] led him to postulate the formation of a "pseudofovea" to account for these findings. Tachistoscopic studies convinced him that the region of clearest vision is not anatomically fixed by the optic pathways but is subject to functional influences. When lesions destroy the old central fovea, a new functional "pseudofovea" serves the purposes of vision. The visual field of the hemianopic patient is not just half of a normal field but undergoes an entire reorganization about the new functional center of clearest vision. As evidence, Fuchs points to the "displacements" which are shown to occur in spatial disorientation. The Axenfeld test is one of the best known; in halving a line, the midpoint does not pass through the center but is displaced toward the side of preserved vision. Fuchs also finds confirmation of spatial reorganization of the field in the observations of Best, who found that patients appeared to be "looking past" the object which they were actually fixing. A simple test was used by Fuchs to show that clearest vision actually shifted from the fixation point. He allowed a patient to fix a given point; directly contiguous was a row of letters. The patient described as the clearest letters not those adjacent to the fixation point, as in normal vision, but those farther removed in the periphery. The reduced sphere of attention is accounted for by actual hemianopia and by reorganization of the field.

Studies in our own case confirm the observation of Fuchs that clearest vision no longer coincides with the fixation point but has been displaced toward the side of preserved vision. We find that there is correct perception of objects in the visual field at the point of clearest vision and to the right, but that forms to the left of clearest vision are misinterpreted. Tests show that relationship of the fixation point to the configuration of the object of attention is of fundamental importance in perception of objects between the fixation point and clearest vision. Symmetric objects are bisected by the direct vision of the patient, so that a W may be seen as a V. When the balance of the configuration is to the left, as E, the entire letter is seen; if the balance is to the right, detached parts to the left, as in the 4 (fig. *3b*), are ignored.

The "overlooking" of parts of the configuration in the left field appears to depend on a complexity of factors. Our studies suggest that in examining a figure the patient's attention is automatically directed so that it centers about a point to the right of the actual fixation point, while the visual object at the fixation point itself has an orienting value. This accounts for the fact that he reads THERE as HERE. The eye is fixed on T, which is seen incompletely, while clear vision begins at H. The patient is able to read to the right from this point

and directs his attention just so far over to the left as to ignore invariably just one or two letters.

The characteristic tenacity with which attention is concentrated on a circumscribed region of the field led Bálint to describe a "mental paresis of gaze" *(Seelenlaehmung des Blickes)*. This limitation of attention results in a perception of details rather than a survey of the total field. The automatic shifting of the eye in reading is impaired and is replaced by jerky ocular movements, which lead to dropping of words and letters, skipping from line to line and consequent dyslexia (Jossmann [3]).

Both the limitation of attention and the deviation of gaze are due in large part to the fact that vision is now a function of only half of the brain. Normal sight, even in one eye, requires the synthetic activity of both hemispheres. Probably the normal visual field represents an equilibrium between two opposing directions of gaze which produce a stereoscopic effect. When one component is missing, the check on the other is lost, and hence the gaze is displaced toward the side of preserved vision. The visual activity of one hemisphere is automatically adjusted to the other, and even in hemianopia the defective field appears in some fashion to attain a functional significance. The patient spontaneously bisects the visual field, as in reading or writing, and possibly has a certain degree of consciousness of the "unseen" field, especially of figures just peripheral to the region of clear vision. Engerth and Hoff [4] have shown that such indistinctly perceived images are of importance in producing hemianopic hallucinations. Poppelreuter demonstrated a tendency to see as complete certain familiar forms, such as circles, which are partly visible in the preserved field but have gaps in their structure that are placed in the defective field. A similar tendency to elaborate given forms was found when our patient was shown a sketch of a face from which the left eye and ear were omitted. Still greater elaboration to the point of confabulation was displayed when the patient readily told the time although he did not know the position of the left hand of the clock.

The "confabulations" are of great interest. We have found that a source of the misinterpretations was in the actual distortion of the visual field. However, a definite psychologic component is also present, probably resulting from a cortical lesion. Anosognosia, a tendency to overlook the existence of a defect, has been reported in many cases of organic disease of the brain. Anton described a hemiplegic patient who was not aware of his paralysis. Sensory aphasia is often accompanied by seem-

3. Jossmann: Dyslexie, Zentralbl. f. d. ges. Neurol. u. Psychiat. **53**:671, 1929.

4. Engerth, G., and Hoff, H.: Ein Fall von Halluzinationen im hemianopischen Gesichtsfeld, Monatschr. f. Psychiat. u. Neurol. **74**:246, 1929.

ing ignorance of the disorder on the part of the patient. Schilder [5] has commented on the frequency with which "body-image" disturbances are associated with lesions of the right hemisphere. In our own review of the literature on spatial disorientation it was noteworthy that this symptom was so frequently found in association with lesions of the right hemisphere, as in the cases of Zutt,[6] Fuchs, Engerth and Hoff, Holmes,[7] Riddoch [8] and Scheller and Seidemann.[9] Possibly the occurrence of aphasia in conjunction with lesions of the left hemisphere obscures the manifestations of spatial agnosia. In any case, this disorder appears superimposed on the ordinary disabilities of homonymous hemianopia. Possibly because there is not merely loss of visual function but also of the representation of that function in the body image, the patient is unable to comprehend that his orientation is defective and therefore cannot adjust himself as well as does the patient with a subcortical lesion of the optic tract. There are elements in the mental picture which our patient presents which are suggestive of a Korsakoff syndrome limited to events in the visual sphere. Attention is poor; there are suggestibility and a flight of ideas in regard to visual objects and a lively imagination enables the patient readily to gloss over his actual inadequacy. This type of mental picture suggests impairment of the function of the brain as a whole with especial involvement of the visual associative centers, perhaps resulting from edema, metabolic disturbances or hyperemia in reaction to the neoplasm.

SUMMARY AND CONCLUSION

A patient with an infiltrating spongioblastoma multiforme in the right temporal lobe and probable extensions into adjacent areas revealed a left homonymous hemianopia and difficulty in spatial orientation. There was a spontaneous tendency to direct visual attention to the right and to ignore objects on the left. Tests showed general constriction of the field of attention and displacement of clearest vision to the right of the fixation point. These disturbances resulted in abnormal perception of visual configurations with consequent concentration on details of a

5. Schilder, P.: The Image and Appearance of the Human Body: Studies in the Constructive Energies of the Psyche, Psyche Monographs, no. 4, London, George Routledge & Sons, Ltd., and Kegan, Paul, Trench, Trubner & Co., Ltd., 1935.

6. Zutt, J.: "Rechts-Linksstörung, konstruktive Apraxie und, reine Agraphie," Monatschr f. Psychiat. u. Neurol. **87**:65, 1933.

7. Holmes, G.: Disturbances of Visual Orientation, Brit. J. Ophth. **2**:449, 1918.

8. Riddoch, G.: Visual Disorientation in Homonymous Half-Fields, Brain **58**:376, 1935.

9. Scheller, H., and Seidemann, H.: Zur Frage der optischräumlichen Agnosie, Monatschr. f. Psychiat. u. Neurol. **81**:97, 1931.

pattern rather than a survey of the total field. Objects at the fixation point were likely to be misinterpreted—a finding which is against a sharp distinction between "object agnosia" and "spatial agnosia." Examinations showed that disorientation in space resulted in dyslexia and reactions such as characterize "simultaneous agnosia" and "constructive apraxia."

A complex problem is presented by the tendency of the patient to "confabulate" about perception of objects toward the side of defective vision. Apparently there is actually a remnant of vision preserved in this field. An important agent in producing the "confabulations" is the disposition of the patient to ignore and cover up his defects, as in pretending to tell time despite inability to see one side of the clock. This tendency is related to body-image disturbances, which appear to occur with particular frequency in association with loss of function of the left side of the body. As in the Korsakoff syndrome, a lively imagination and lack of critical attention obscure reality and make it possible for the patient to avoid a realization of his inadequacy. The mental picture and spatial disorientation appear to be functional disturbances which result from cortical destruction and complicate the adjustment to homonymous defects of the visual field.

THE HUGHES PROCEDURE FOR REBUILDING A LOWER LID

SANFORD R. GIFFORD, M.D.
CHICAGO

In 1937 Hughes[1] described a procedure in which the tarsus and conjunctiva of the upper lid are employed to replace part or all of the lower lid. Use of one lid to replace another had been employed as long ago as 1881, when Landolt[2] described his method. It was devised to produce firmer adhesion between the two lids than had been obtained by earlier methods. Landolt's object was to obtain skin to cover defects in the lid in which a portion of conjunctiva remained. Hence for defects in the lower lid he buried a flap of conjunctiva from the lower lid between the two layers of the normal upper lid, freshened the border of the lid and sutured it to the edge of the defect. After several months, when horizontal folds in the skin showed that traction on it had been relieved by stretching, a new fissure was made above the border of the upper lashes, which was now transplanted to the lower lid. No lashes were, of course, present in the new border of the upper lid. The procedure was used for defects of either lid and even for defects of both lids. An example of the latter condition was described in 1885[3] in which half of the upper lid, all that remained of both lids, was made into an upper and a lower lid. Modifications of this procedure were described by Czermak and Kuhnt, while Cirincione and Löwenstein[4] sutured the remaining mucosa of the affected lid to that of the normal lid, the defect in the skin being replaced by free grafts.

Dupuy-Dutemps also appreciated the value of employing tarsus and conjunctiva from the upper lid in defects involving these structures. His method, as described by Bourguet,[5] consists in splitting the upper lid and uniting its conjunctivotarsal layer to the conjunctiva below. The two edges of skin were also sutured, but after two weeks the sutures were removed, allowing the skin of the upper lid to retract. At this time a skin flap with rotated pedicle was made from the upper lid and applied

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From the Department of Ophthalmology, Northwestern University Medical School, and the Cook County Hospital.

1. Hughes, W. L.: New Method for Rebuilding the Lower Lid: Report of a Case, Arch. Ophth. **17**:1008 (June) 1937.

2. Landolt: Arch. d'opht. **1**:9, 1881.

3. Landolt: Arch. d'opht. **5**:481, 1885.

4. Czermak, Kuhnt, Cirincione and Löwenstein, cited in Axenfeld, T., and Elschnig, A.: Handbuch der gesamten Augenheilkunde, Berlin, Julius Springer, 1922, pp. 356-358.

5. Bourguet: Rev. de chir. structive **7**:91, 1937.

to the denuded surface of the tarsus for the new lower lid. The procedure of Dupuy-Dutemps has been described in English by Ferris Smith,[6] who reproduced the original illustrations. De Saint-Martin [7] employed a method even more like that of Hughes. His first step is like that of Dupuy-Dutemps, but a flap of skin from the cheek is made after the Hungarian method and rotated so as to come into apposition with the border of the upper lid without tension. This method, or a similar one, was apparently employed by Imré and Blaskovics.

The Hughes procedure was especially designed for cases in which all or nearly all of the tarsus and conjunctiva of the lower lid, as well as the skin, must be sacrificed. Hughes recognized that if blepharoplasty is performed at the time of operation for a neoplasm, the skin of the face can be mobilized so that it will cover the defect if a suitable bed of tarsus, lined with conjunctiva, can be provided. His procedure, while superficially similar, is really the reverse of the Landolt procedure, since it transfers part of the tarsus and conjunctiva of the upper lid to the new lower lid, no skin from the upper lid being sacrificed and its border of lashes, much more important cosmetically than the lower lashes, being left intact. The advantage of implanting skin-bearing hair follicles (stage 2) while the borders of the lids are fused is obvious, since implanting such grafts in a free border is exceedingly difficult. No provision for providing new lashes for either lid was described by Landolt.

Experience with 3 cases in which the Hughes method was employed has brought out certain minor complications which may be obviated by a few technical details not mentioned in Hughes' article.

Case 1.—Ben H., aged 54, had received treatment with radium for carcinoma of the lower lid during the past three years. The growth had progressed since the last irradiation. On Feb. 18, 1938, the Hughes procedure was employed, the whole lower lid being excised to the level of apparently healthy tissue. The upper lid was split, its epithelial border was excised with care to spare the lash follicles, and its mucosa was sutured to the edge of bulbar conjunctiva remaining after excision of the lower lid. Three double-armed sutures were inserted deeply in the tarsus midway between its upper and its lower border and brought out through the skin of the face, which had been dissected freely. The sutures were placed far enough from the upper border of the lower skin flap so that the cut edge of skin fell into place without tension against the denuded cutaneous border of the upper lid, where it was secured by a second row of sutures. A small opening for drainage was left at the outer angle leading into the conjunctival sac. The tear point and lower canaliculus were excised, as the skin over them was involved in the neoplasm.

6. Smith, F.: Reconstructive Surgery of the Head and Neck, in Nelson Loose-Leaf System, New York, Thomas Nelson & Sons, 1928, p. 161.

7. de Saint-Martin: Bull. Soc. d'opht. de Paris **49**:36, 1937; abstracted. Zentralbl. f. d. ges. Ophth. **39**:389, 1937.

At the first dressing a rather marked swelling of the lids was present, which closed the outer opening so that no drainage of the sac was available. The small opening was enlarged, and a thick purulent secretion was seen in the sac, which could be removed only in part. It was necessary to enlarge this opening further several times, in spite of which the profuse discharge continued until the lids were separated eight weeks after the primary operation. Because of the secretion, no attempt was made to implant hair follicles for the border of the lower lid. Figure 1 *A* shows the condition five weeks after the first operation. When the lids were separated, a peculiar condition was found which seemed to account for the profuse discharge. Along the line where the two layers of conjunctiva were united by silk sutures tied inside the conjunctival sac four polypoid masses were found, the largest measuring 1 cm. in diameter. When these were snipped off, the discharge rapidly disappeared.

Hughes had not stated in his article whether the skin and conjunctiva of the new borders of the lids were united by sutures. It was thought best to place such sutures in this case, but this proved to be a mistake. The shortened conjunctiva drew the border of the lashes up in such a way as to produce a troublesome trichiasis, requiring separation of the two layers five weeks after the lids were opened, the large raw surface being covered by mucous membrane from the lip.

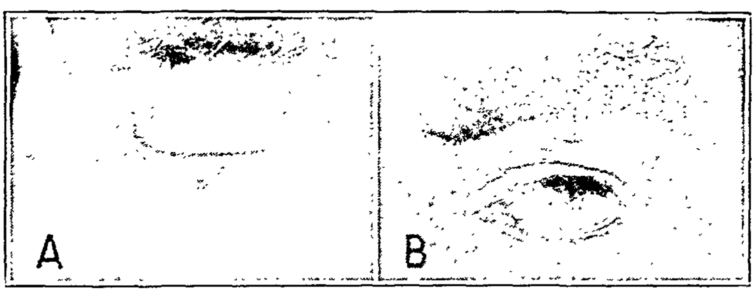

Fig. 1 (case 1).—*A,* results five weeks after the first stage of the operation; *B,* results four months after the first operation.

Figure 1 *B* shows the result four months after the first operation. The lower lid is somewhat short and stands slightly away from the globe, but its position is slowly improving. The lashes of the upper lid fell out but are now growing back. The trichiasis is giving no more trouble. The cornea, which suffered no damage while the lids were closed, developed a few erosions after the lids were opened, but these healed rapidly. At the time of writing, there are no signs of recurrence of the neoplasm. The patient cannot completely close the lids without an effort, but the cornea is sufficiently protected.

Case 2.—Ben G., aged 28, was seen with a rapidly growing mass on the left lower lid (fig. 2 *A*), which had been noted for only two months. The patient was practically blind from simple atrophy of the optic nerve. The Wassermann reaction of the spinal fluid was positive; hence an infectious granuloma of syphilitic or other origin was suspected. Biopsy, however, showed the growth to be a basal cell carcinoma. The Hughes procedure was performed on March 4, 1938, only the outer two thirds of the lower lid being excised, the outer two thirds of the upper lid being employed to replace it. The inner third of the palpebral fissure was left open for drainage. Six weeks after the first operation a strip of skin from the lower nasal border of the brow bearing from eight to ten lash follicles was implanted in a groove made below the upper lash follicles. It healed in place but after several weeks became somewhat thin and dry.

The lids were separated on May 20, twelve weeks after the first operation. Although sutures were placed only at the inner and the outer angle of the wound, the lashes were somewhat inverted by contraction of the conjunctiva. This will probably require an implant of mucosa from the lip. The cosmetic effect, however, is good (fig. 2 *B*).

CASE 3.— George, aged 55, was first seen in January 1936. After surgical removal of a carcinoma from the lower lid four years before, with recurrence, he had received intensive treatment with radium. The result was complete loss of the lid, the skin being extremely thin and tightly adherent to the bone at the lower orbital margin. Two operations were performed during 1936 in an attempt to replace the lower lid with a pedicled flap previously lined with a Thiersch graft. The nutrition of the lower edge of the graft, which rested on the bony orbit, was insufficient, however, and the graft sloughed, leaving the

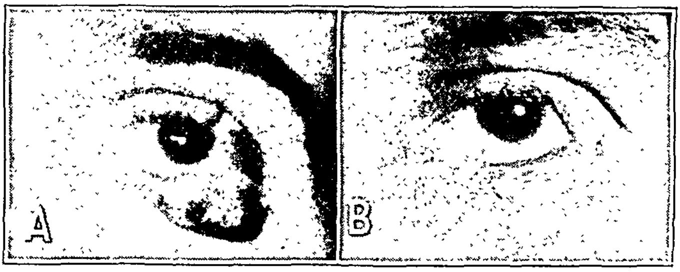

Fig. 2 (case 2).—*A,* eye before operation; *B,* results three months after the first operation.

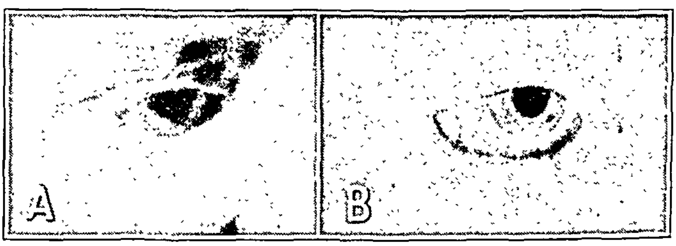

Fig. 3 (case 3).—*A,* eye before the Hughes operation; *B,* result after the last operation.

condition about as before, except that a strip of skin from the upper lid had been sacrificed (fig. 3 *A*). On Feb. 4, 1938, the first stage of the Hughes procedure was performed. Owing to the extreme thinness of the skin, it seemed doubtful if it could be made to heal over the tarsus of the upper lid. The advisability was considered of releasing tension on the skin of the lower lid by fashioning a triangular flap of skin and soft parts after the Hungarian method, with the incision along the nasal border of the cheek. After free dissection of the skin, however, it came into place with little tension, and the additional incisions were not made. On March 25, a strip of skin from the temporal border of the scalp bearing from fifteen to twenty hair follicles was implanted. Skin from this location was used instead of from the brow, where the hair was already very thin, and the implant was made, not into the line of the incision, but 6 mm. above it, since it was evident that this amount of skin from the upper lid must be added to the lower lid on account of the extreme deficiency of skin. The graft healed, with most of the hair follicles surviving.

On April 22 an incision was made through skin, tarsus and mucosa just below the implant of skin. The incision was made only 14 mm. long, leaving the new fissure somewhat short at the nasal side as a precaution against contraction of the lower flap. Only two sutures were placed between the skin and the mucosa near either angle. The lower lid was somewhat short, and the new fissure had a bizarre shape, since the upper lid had lost skin by the previous operations and the present one.

On May 27 an incision was made in the upper lid, and the outer portion of the levator muscle was cut, so that the lid could be pulled down to the lower lid by sutures. The defect was covered by a Thiersch graft. This allowed the upper lid to come down into a fairly normal position, and on June 17 the inner angle of the palpebral fissure was opened further, so as to make the fissure as nearly as possible the same length as the opposite one (fig. 3 *B*).

COMMENT

A personal communication from Dr. Hughes and a view of the film which he sent have made clear a number of points which were not clear to me from his article. Hughes stated that slight trichiasis occurred in only 2 of his 20 cases. This may be attributed to three details in procedure. First, the dissection of the tarsus and the levator from the skin of the upper lid must be complete, so that the shortened tarsus is not drawn up by adhesions in such a way as to invert the border of the lid. Second, a line of deep, double-armed sutures is placed in the tarsus and brought out through the skin of the upper lid about 4 mm. above its border. This is in addition to the similar line of sutures which holds the skin of the new lower lid to the tarsus. In the film the use of a subcuticular suture to unite the borders of the two lids is shown in place of interrupted sutures. Third, the graft of hair-bearing skin for the border of the lower lashes is made quite large, and this skin which is left between the two lines of lashes probably aids in preventing contraction of the border of the upper lashes. No sutures are employed in stage 3 when the palpebral fissure is opened, the lashes being smoothed back into place with a thick ointment and held in this position by a pressure dressing during healing.

Hughes had no trouble with retained tears or secretion, though he left only one small opening at the inner or outer angle for drainage.

CONCLUSIONS

1. The need for adequate drainage is illustrated in the first case. Evidently the retained secretion promoted granulation tissue along the suture line between the two layers of mucosa, and the granulation tissue promoted still more secretion. It would seem best, when possible, to leave openings at both the inner and the outer angle, so that through and through irrigation could be performed.

2. Uniting the layers of the new borders of the lids by sutures is unnecessary and should be omitted, as it is likely to cause entropion. If entropion does occur, it can be efficiently corrected by a graft of mucosa from the lip.

3. The advantages of employing the procedure as a primary operation at the time the neoplasm is removed are illustrated by the inferior result in case 3, in which much skin had been lost by radium, contraction and previous operations.

4. When the condition seen in case 3 is already present, it would be best to fashion a flap according to the Hungarian method, as employed by de Saint-Martin, so that no skin from the upper lid would need to be sacrificed. It might also be advisable to delay separation of the lids for one or two months longer than was done in this case, so that contraction would be minimized.

5. The Hughes procedure is an exceedingly useful method for rebuilding all or part of a lower lid when both skin and conjunctiva must be removed with the neoplasm. The results are certainly better than could be obtained with pedicled grafts, the edges of which are nearly always unsightly. Pedicled grafts are likely to be impracticable when a lining of mucous membrane is required.

LOCALIZING VALUE OF INCONGRUITY IN DEFECTS IN THE VISUAL FIELDS

DAVID O. HARRINGTON, M.D.
SAN FRANCISCO

Asymmetry or incongruity of incomplete homonymous hemianopic defects in the visual fields is a constant finding with lesions of the anterior portion of the geniculocalcarine pathway. It becomes less and less evident the farther back the visual pathway is interrupted, until in the defects in the visual fields, found in association with lesions of the occipital lobe, the degree of symmetry is very striking.

The value of perimetry in the lateral localization of tumors of the brain has long been known. The study of this phenomenon of varying incongruity, both in the literature and in the following case reports, has shown it to be of definite value in localizing cerebral lesions anteroposteriorly as well as laterally.

If one carefully examines the literature relative to this clinical entity, analyzing the defects in the visual fields described in the various articles and collecting those charts of the visual fields illustrative of proved lesions of the temporal, parietal or occipital lobe, one is immediately struck by the following facts:

1. Quantitative perimetry is the only method of real value in detecting the finer degrees of asymmetry in these incomplete homonymous hemianopic defects in the visual fields. A sufficient number of test objects must be used at various distances to prevent the possible accusation of poor technic as the cause of the incongruity. The full extent of the asymmetry may be brought out only by testing three, four or five isopters.

2. When this method is used and the resultant charts of the visual fields are complete and accurate in their data, lesions of the temporal, parietal and occipital lobes of the brain are seen to produce homonymous hemianopias, which when incomplete show varying degrees of asymmetry constant enough to be of localizing value.

This incongruity has been noted and extensively commented on by Henschen,[1] Rönne,[2] A. Meyer,[3] Schirmer,[4] Wilbrand and Saenger,[5]

Read before the Society of Neurological Surgeons, San Francisco, June 9, 1938.

From the Division of Ophthalmology, Department of Surgery, University of California Medical School.

1. Henschen, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1935-1936, vol. 1, p. 903; vol. 3, p. 773.

(Footnotes continued on next page)

Cushing,[6] Traquair,[7] Horrax and Putnam,[8] Oldberg,[9] Peter[10] and many others.

In the past, however, relatively little clinical significance has been attached to these incongruous defects, the majority of articles on the subject being concerned primarily with the anatomic explanation of the phenomenon. Cushing mentioned upper homonymous quadrantanopias as characteristic of the defects in the visual fields produced by lesions of the temporal lobe, and Traquair, commenting on Cushing's cases, attributed the incongruity to interference with the optic tract and definitely stated that its presence localizes the lesion in the subgeniculate pathway. Horrax and Putnam called attention to the greater tendency to symmetry of defects in the visual fields in cases of lesions of the occipital lobe but attached more localizing value to the macular sparing which they commonly found.

In the past few years I have quantitatively examined the visual fields of most of those patients in the neurosurgical service of Dr. Howard Naffziger, where initial tests, usually done by the intern or assistant resident, have demonstrated the presence of homonymous hemianopia.

It has been my practice in the majority of instances to conduct the perimetric examination without a foreknowledge of the other neurologic findings, and I have deliberately attempted, when possible, to localize the lesion by perimetric findings alone.

Only those cases in which the site of the lesion was subsequently proved at operation or at autopsy have been used in this series. Hemianopias which were complete to all test objects were obviously of no value in this study, though these are increasingly infrequent due

2. Rönne, H.: Ueber die Inkongruenz und Asymmetrie im homonym hemianopischen Gesichtsfeld, Klin. Monatsbl. f. Augenh. **19**:399, 1915.

3. Meyer, A.: The Connections of the Occipital Lobes and the Present Status of the Cerebral Visual Affections, Tr. A. Am. Physicians **22**:7, 1907.

4. Schirmer, O.: A Case of Incongruous Homonymous Hemianopsia, Arch. Ophth. **41**:136, 1912.

5. Wilbrand, H., and Saenger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1917, vol. 7, p. 152.

6. Cushing, H.: The Field Defects Produced by Temporal Lobe Lesions, Brain **44**:341, 1921.

7. Traquair, H. M.: An Introduction to Clinical Perimetry, ed. 3, St. Louis, C. V. Mosby Company, 1938, pp. 60, 242 and 253-255.

8. Horrax, G., and Putnam, T. J.: Distortions of the Visual Fields in Cases of Brain Tumor, Brain **55**:499, 1932.

9. Oldberg, S.: An Attempt to Explain the Quadrantanopsia in Tumor of the Temporal Lobe, Acta med. Scandinav. **93**:330, 1937; abstracted, Surg., Gynec. & Obst. **66**:326, 1938.

10. Peter, L.: The Principles and Practice of Perimetry, ed. 2, Philadelphia, Lea & Febiger, 1923, p. 225.

to the earlier diagnosis of tumors of the brain. Lastly, those cases were discarded in which it was felt that lack of cooperation or too rapid fatigue gave unreliable results. With patience and repeated short examinations, this reason for failure has been of minor importance.

In many instances, to avoid confusion, only those isopters have been charted which give the needed pertinent information. In every case, however, the hemianopic border was examined with at least four test objects at two distances.

The following case histories, with charts of the visual fields, serve to demonstrate the constancy of the incongruity in defects in the visual fields when the temporal lobe is involved. In marked contrast is the absolute symmetry, extending even to minor irregularities, in the incomplete homonymous hemianopic defects found in cases of proved lesions of the occipital lobes.

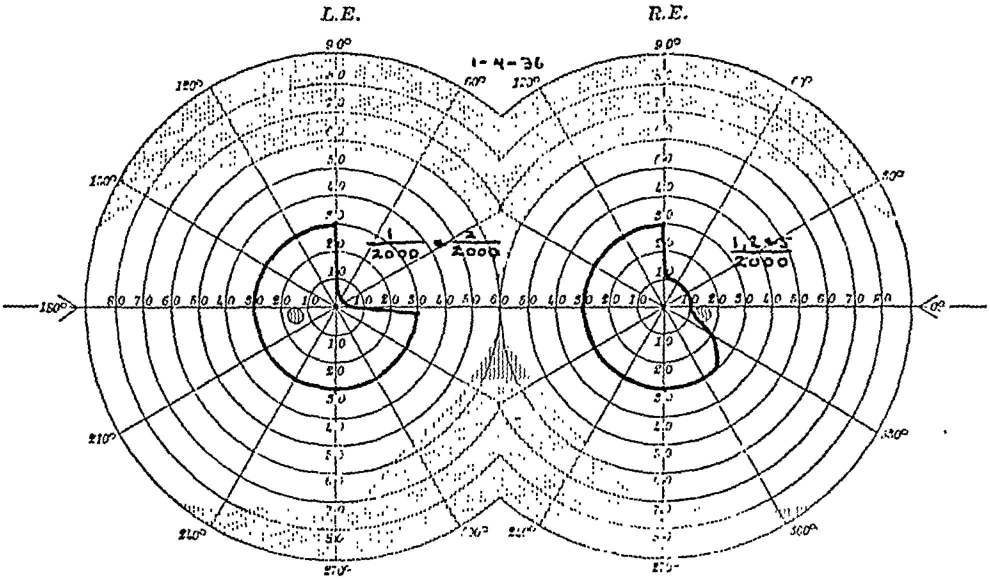

Chart 1 (case 1).—Visual fields of W. K.

LESIONS OF THE TEMPORAL LOBE

Case 1.—W. K., a 34 year old man, had a large glioma of the left temporal lobe. Partial removal was accomplished. Chart 1 shows the incongruous right upper quadrantanopia found before operation.

Case 2.—S. W., a 58 year old man, whose chief complaints were headache, hallucinations of smell and blurred vision, had a hemianopic defect in the visual fields. Operation in March 1934 exposed a spongioblastoma of the right temporal lobe, which was partially removed. Five months later there was a recurrence of symptoms, with a swelling of the area of decompression. Quantitative examination of the fields showed the incongruous left upper quadrantanopia shown in chart 2.

Case 3.—F. S., a 36 year old woman, had attacks of jacksonian epilepsy on the right side. The entire neurologic examination gave negative results except

for the incongruous defects in the visual fields shown in chart 3. There was no choking of the optic disks. At operation practically the entire left temporal lobe and part of the parietal lobe were removed, most of the middle fossa on that side being exposed. The glioma weighed 150 Gm., The optic tract was seen but was undamaged. Postoperative visual fields showed complete hemianopia for all test objects.

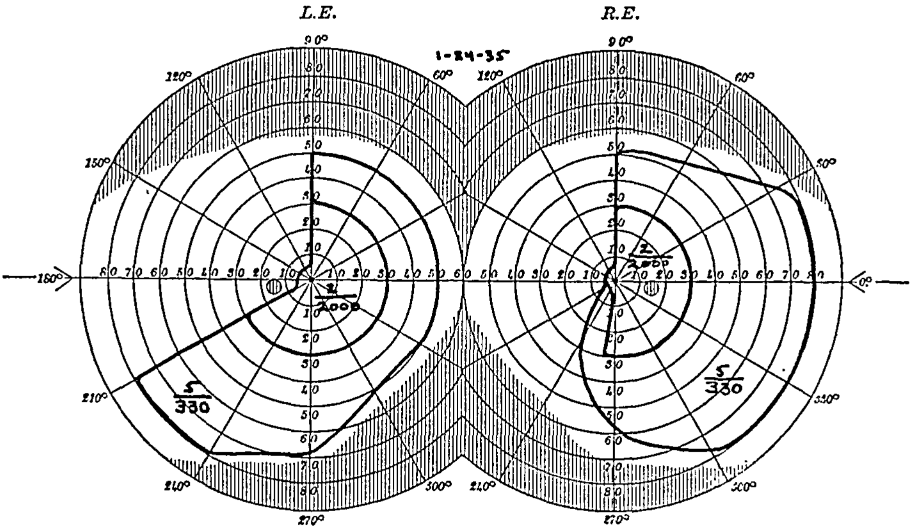

Chart 2 (case 2).—Visual fields of S. W.

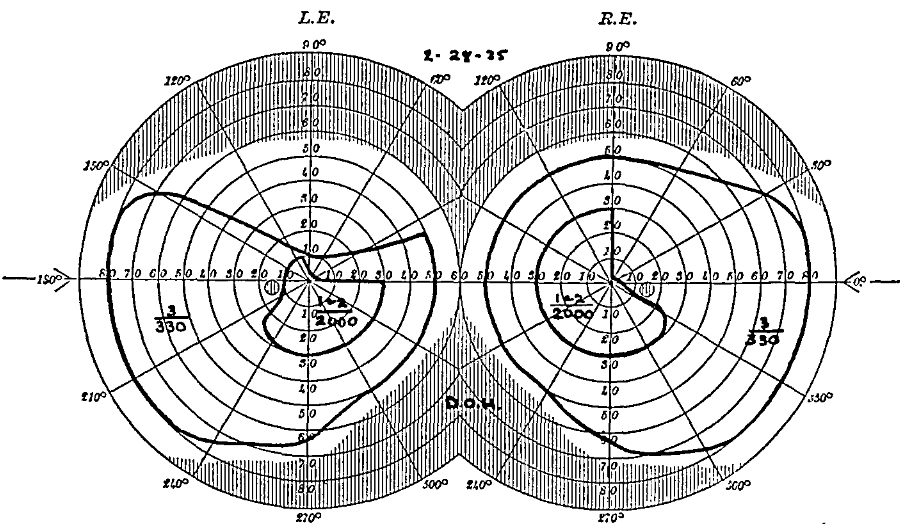

Chart 3 (case 3).—Visual fields of F. S.

Case 4.—W. M., a 35 year old man, on operation was found to have a large, deep-seated glioma in the right temporal lobe. Chart 4 shows the incongruity in the left upper homonymous quadrantanopia, noticeable with all test objects but most obvious with the larger visual angles.

Case 5.—A. V., a 37 year old woman, suffered from headache, loss of vision, in the left eye, failing memory and speech difficulties. Examination showed bilateral papilledema, weakness of the right side of the face and the right arm and a right homonymous hemianopia. At operation a large dural endothelioma

attached to the left petrous portion of the temporal bone and invading the temporal lobe was almost completely removed. Chart 5 shows the asymmetric homonymous hemianopia, more striking in the postoperative fields.

Case 6.—R. G., a 46 year old man, entered the hospital with a history of convulsive seizures and loss of vision. Chart 6 shows the marked incongruity of the

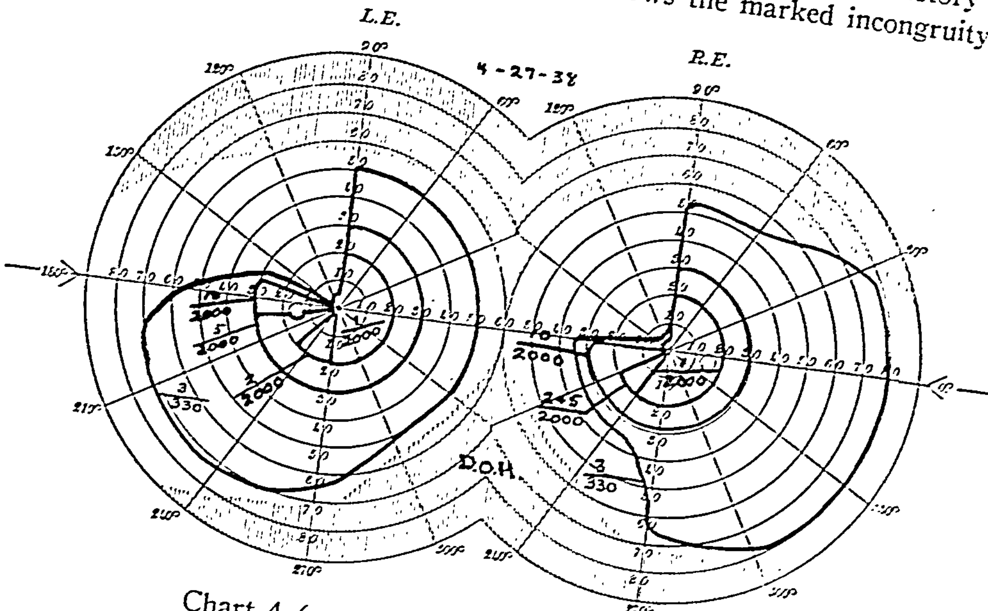

Chart 4 (case 4).—Visual fields of W. M.

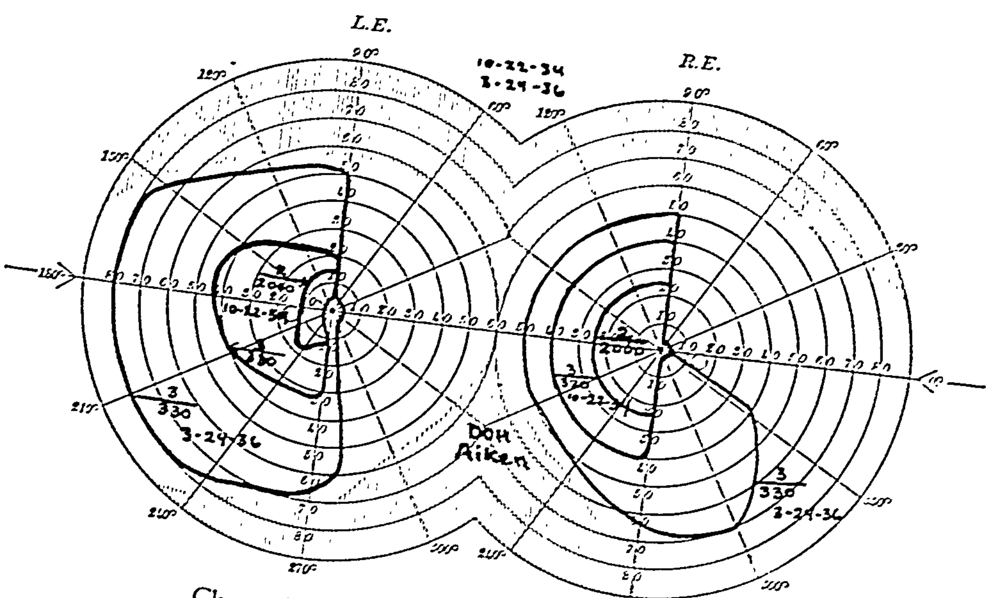

Chart 5 (case 5).—Visual fields of A. V.

left upper homonymous quadrantanopia. A large spongioblastoma was removed from the right temporal lobe.

Case 7.—J. F., a 35 year old man, when first seen in 1934 had headache and convulsive attacks of five months' duration. Neurologic examination gave essentially negative results. In 1936 reexamination showed a facial weakness on the

left side; the convulsive seizures were more frequent and uncinate-like and involved mainly the left side. Quantitative perimetric studies gave negative results. Roentgenograms showed marked displacement of the midline to the left.

At operation a large portion of a huge astrocytoma of the temporal lobe was removed. It extended from the tip backward about 6 cm.

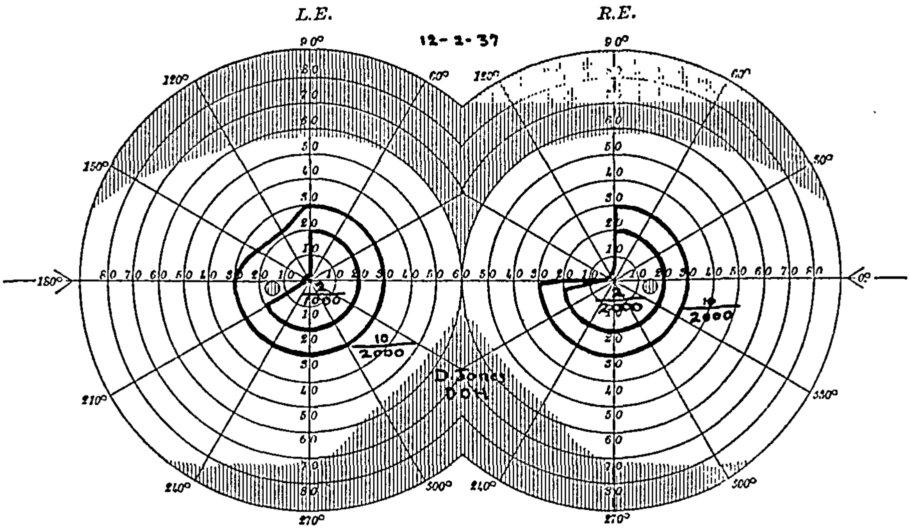

Chart 6 (case 6).—Visual fields of R. G.

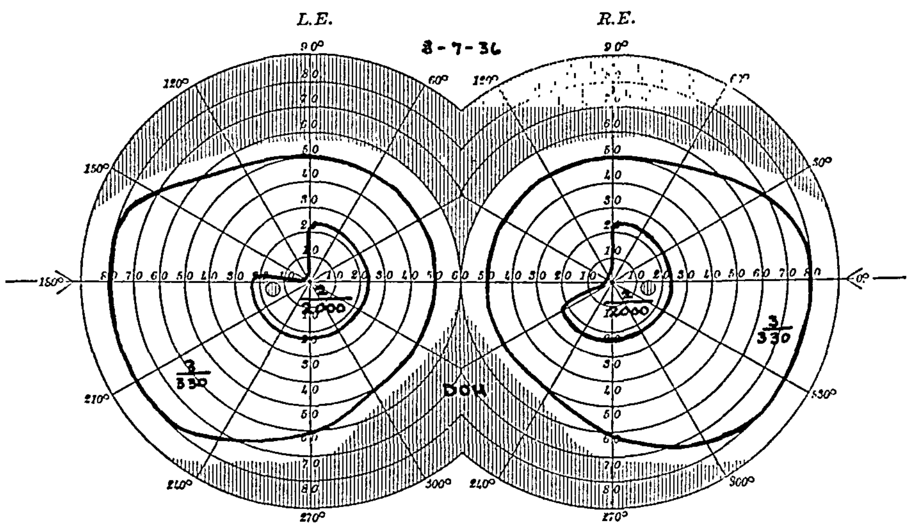

Chart 7 (case 7).—Visual fields of J. F.

Perimetric fields taken two months later (chart 7) showed an incongruous left upper homonymous quadrantanopia detected only with small visual angles.

CASE 8.—W. F., a 26 year old man, had chronic otitis media on the left side which developed into chronic mastoiditis and subsequently into an abscess of the left temporal lobe. Radical mastoidectomy was performed, followed by drainage

of the abscess, with eventual recovery. The visual fields shown in chart 8 are those taken four months after operation, showing a definite incongruous right homonymous quadrantanopia for small visual angles. There was no change one year after operation.

Charts of the visual fields similar to or almost identical with charts 1 to 8 will be found scattered throughout the literature wherever the function of the temporal lobe is under investigation. The most notable examples, however, are those seen in Cushing's[6] article on lesions of the temporal lobe. With all the requirements of careful quantitative perimetry fulfilled, his charts demonstrate, in a striking manner, the incongruity of the incomplete hemianopias found in lesions of the anterior portion of the suprageniculate visual pathway.

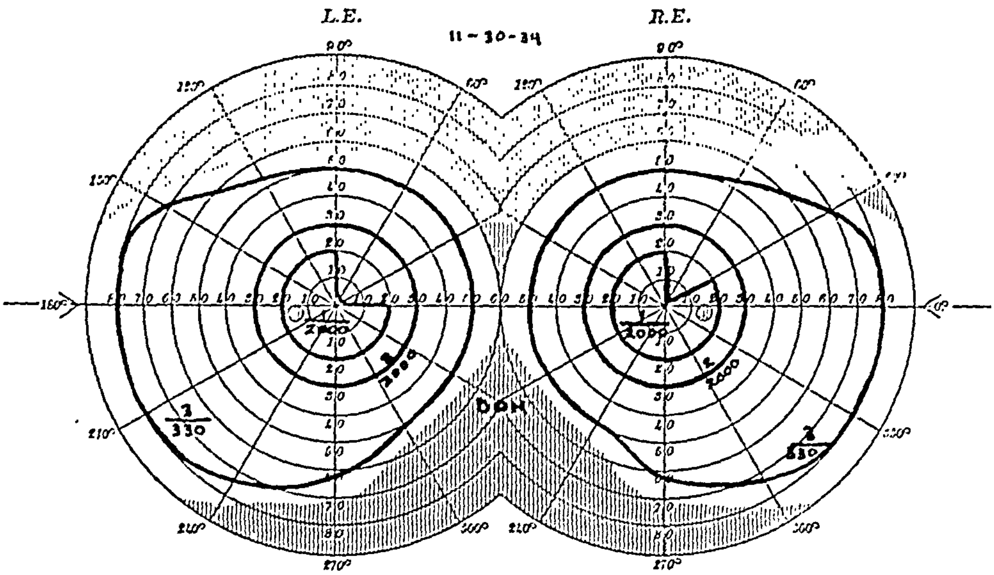

Chart 8 (case 8).—Visual fields of W. F.

LESIONS OF THE PARIETAL LOBE

Lesions involving this area of the brain alone, without extension either forward or backward, are extremely rare. Of 4 patients with such lesions who were examined by me, 2 were too ill to give reliable answers during examination and the other 2 had extremely extensive damage to the brain, almost certainly encroaching on the occipital cortical area. Complete symmetry of a homonymous hemianopia was present in 1 of the latter 2, while in the other the asymmetry was so slight as to be open to question.

Traquair[7] in his textbook illustrated the slight asymmetry which may be found by quantitative perimetric study in cases of lesions of the parietal lobe. He attributed the incongruity to the sloping edge of the defect. His figures 203, 205 and 206 are worthy of careful study.

LESIONS OF THE OCCIPITAL LOBE

CASE 9.—B. R., a 19 year old youth, noted a sudden visual loss following a blow to the occiput. Perimetric study showed a congruous, incomplete left homonymous hemianopia, which persisted after the right occipital lobe was exposed and needled and a quantity of old hemorrhage removed. Chart 9 shows the symmetry of the defect as found with the larger visual angles.

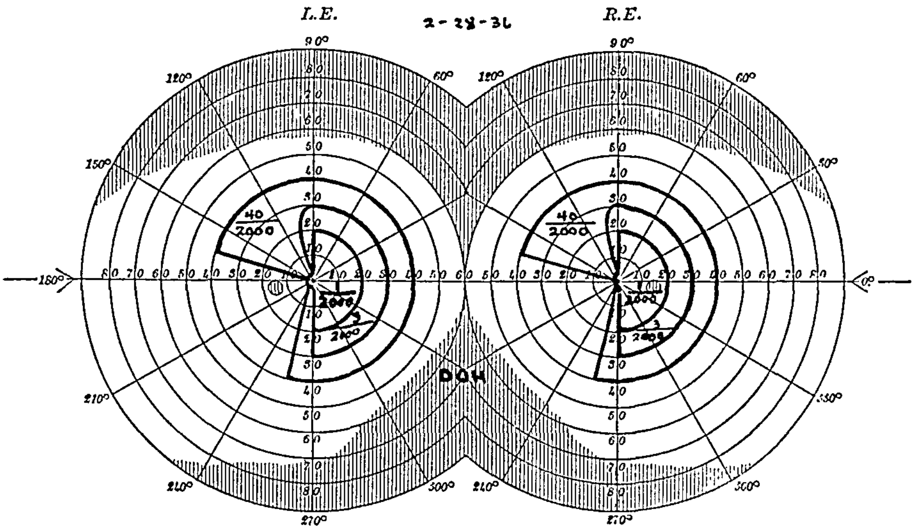

Chart 9 (case 9).—Visual fields of B. R.

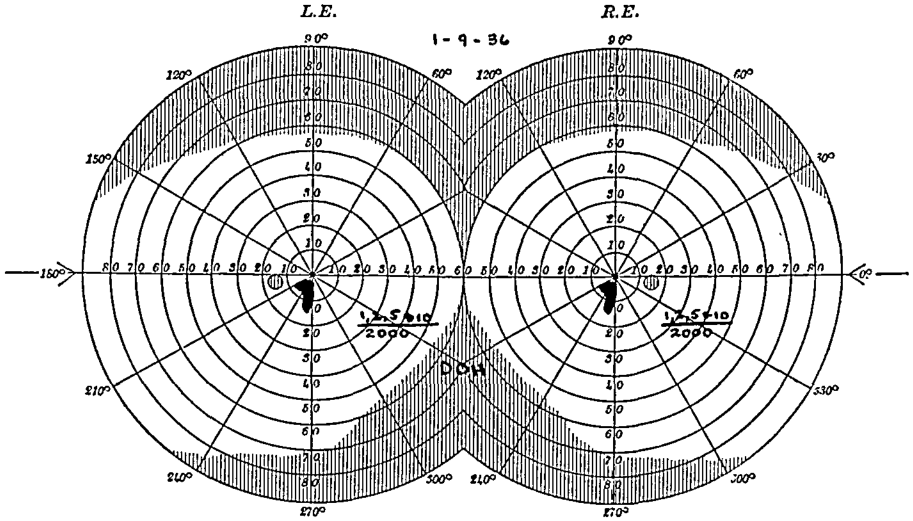

Chart 10 (case 10).—Visual fields of C. C.

CASE 10.—C. C., a 25 year old man, had post-traumatic epilepsy due to a bullet wound in the right upper temporal area. Roentgenograms localized the small lead bullet lying about ¼ inch (0.6 cm.) from the extreme upper end of the right occipital bone. The complete congruity of the irregular homonymous scotoma seen in chart 10 is striking. The injury was probably a minute one in the upper lip of the calcarine fissure at the right occipital pole.

Further excellent examples of this striking symmetry of the defects in the two fields in lesions in the occipital cortex are found in the articles of Holmes and Lister [11] and of Horrax and Putnam.[8] An outstanding example of the manner in which this congruity is carried into the most minute irregularities is seen in Traquair's figure 223.

From a study of charts 1 to 10 and those of other authors, it will be seen that perimetric examination is of definite assistance to the ophthalmologist, neurologist and neurosurgeon not only as a means of determining the right or left sidedness of a lesion of the brain but also in localizing it in a dorsoventral direction. When the visual fields are routinely examined in all cases in which a disease of the brain is suspected, there will be a high percentage of cases with interruption of the visual pathway and resulting defects in the visual fields. All that is needed to make this test of more accurate localizing value is the careful and general use of a standardized and accurate method of quantitative perimetry.

Now, when one comes to the anatomic explanation of this phenomenon of incongruity of defects in the visual fields in incomplete homonymous hemianopias, one enters a realm of great diversity of opinion.

Wilbrand [12] expressed the belief that the fibers of the visual pathway were diffusely scattered throughout the cerebrum and that this irregularity accounted for the irregularity in the defects in the visual field.

Rönne,[2] apparently greatly against his will, was forced to admit that in certain cases this dispersion must exist to explain certain incongruous defects which he found. He expressed the opinion, however, that they were the exception to the rule and that the asymmetry is for the most part small in extent and limited to the borderline between both fields.

Cushing [6] predicated his explanation of the existence of asymmetric quadrantanopias in cases of tumor of the temporal lobe on his belief in the existence of Meyer's [3] temporal loop consisting of three divisions of the optic radiation, dorsal, lateral and ventral, and that while the dorsal and lateral bundles are rather direct in their course from the external geniculate body to the calcarine cortex, the ventral bundle makes a wide forward and lateral detour around the temporal horn of the lateral ventricle before running back to turn around the posterior end of the ventricle into the calcarine cortex. While he felt that the loss of the visual field was due to direct pressure on the radiation, he did not comment on the cause of the asymmetry.

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11. Holmes, G., and Lister, W. T.: Disturbances of Vision from Cerebral Lesions, Brain **39**:34, 1916.

12. Wilbrand, H.: Ueber die wissenschaftliche Bedeutung der Kongruenz und Inkongruenz der Gesichtsfelddefekte, J. f. Psychol. u. Neurol. **40**:133, 1930. Wilbrand and Saenger.[5]

Traquair [13] and Kravitz [14] expressed doubts as to the existence of Meyer's loop and explained the incongruity on the basis of pressure on the optic tracts from the closely overlying temporal lobe. Traquair, in fact, definitely stated that an asymmetric hemianopia is indicative of interference with the subgeniculate pathway.

Oldberg [9] in a recent paper rather ingeniously ascribed the quadrantanopias found in cases of lesions of the temporal lobe to disturbance in nutrition in the external geniculate body due to pressure of the temporal lobe on a nutrient artery, the anterior choroid. According to him, this artery, supplying the lateral part of the lateral geniculate body, is wedged, in the greater part of its course, between the optic tract and the temporal lobe and is subject to varying degrees of pressure by expanding lesions. He stated that he does not believe that a direct pressure effect on the tract will explain these quadrant defects in the visual fields, as the pressure would have to be selectively localized on the cross-section half of the optic tract. He also disagreed with the popular hypothesis that the pressure effect is on the optic radiation, stating that it is "not supported by clinical observation and is contradicted by many."

Peter [10] admitted that one explanation for asymmetry in homonymous hemianopias could be found in the "dissociation of the nerve fibers from corresponding retinal points from chiasm to cortical centers." Though he considered this explanation of "much anatomic interest," he preferred to explain the incongruities as due either to "poor technic" or "to the difference in relative sensitivity of corresponding retinal points and the various parts of the retina."

Putnam [15] in a series of papers in 1926 concluded on the basis of purely anatomic observations in animals and in man that different bundles of fibers must keep their relative positions accurately in the optic radiation, but adjacent fibers in the radiation do not necessarily supply closely adjacent points in the cortex. He stated that projection fibers proceeding from homologous points in the two retinas are farthest apart at the geniculate body and gradually approach one another as they draw near the visual cortex. Putnam had noted the incongruity in defects in

13. Traquair, H. M.: The Course of the Geniculo-Calcarine Visual Path in Relation to the Temporal Lobe, Brit. J. Ophth. **6**:251, 1922.

14. Kravitz, D.: Quadrant Defects in Temporal Lobe Tumors, Am. J. Ophth. **14**:781, 1931.

15. Putnam, T. J., and Putnam, I. K.: Studies on the Central Visual System: I. The Anatomic Projection of the Retinal Quadrants on the Striate Cortex of the Rabbit, Arch. Neurol. & Psychiat. **16**:1 (July) 1926. Putnam, T. J.: II. A Comparative Study of the Form of the Geniculostriate Visual System of Mammals, ibid. **16**:285 (Sept.) 1926; III. The General Relationships Between the Extra Geniculate Body, Optic Radiation and Visual Cortex in Man, ibid. **16**:566 (Nov.) 1926; IV. The Details of the Organization of the Geniculostriate System in Man, ibid. **16**:683 (Dec.) 1926.

the visual fields in cases of injury to the anterior portion of the optic radiation as compared to the superposable defects found in cases of lesions of the posterior radiation or cortex. He expressed the belief that this was supporting evidence that there was a shifting of fibers in the fasciculus, and he called attention to Minkowski's [16] like opinion, reached by a different path.

In 1935, in a paper read before the Western Ophthalmologic Society,[17] I gave clinical reasons for believing that the incongruity found in hemianopic defects in the visual fields in cases of lesions of the temporal lobe could best be explained by the dissociation in the temporal lobe of fibers from corresponding retinal points and, furthermore, that this separation of homologous fibers persisted in lessening degree throughout the optic radiations, as far posterior as the postparietal area. I believe that further case studies confirm this view.

The clinical observations which substantiate this theory follow:

1. The regularity, both in the literature and in my own few cases, with which lesions of the temporal lobe produce markedly asymmetric defects in the visual field, while the more posterior the lesion is found the more symmetric becomes the loss in the visual fields.

2. The production of these same characteristics of the visual fields in cases of nonexpanding, relatively nonpressure-producing lesions, such as hemorrage, injuries and abscess.

3. The occasional persistence of similar or identical defects in the visual fields after operative removal of their contributory lesion.

4. Cases in which removal of large enough portions of the temporal lobe has exposed an undamaged optic tract and left a homonymous hemianopia.

5. Cases of incongruous hemianopia in which the greatest defect is contralateral to the lesion, thus refuting the theory of varying retinal sensitivity.

6. Even those cases in which there is a rapid restoration of the normal field after operative removal of the causal lesion. It has been argued that these are the cases which are the strongest evidence in favor of the theory of Traquair of pressure on the optic tract. But Meyer showed that the fibers of the optic radiation were resistant to destruction and survived as long as their beginning and end were intact. Direct

16. Minkowski, M.: Ueber den Verlauf, die Endigung und die zentrale Repräsentation von gekreuzten und ungekreuzten Sehnervenfasern bei einigen Säugetieren und beim Menschen, Schweiz. Arch. f. Neurol. u. Psychiat. **6**:201, 1920; **7**:268, 1920.

17. Harrington, D. O.: The Optic Radiations in the Temporal Lobe, Tr. West. Ophth. Soc. **2**:131, 1935-1936.

pressure on these fibers, which is much easier to explain than pressure on the optic tract, might therefore only temporarily interrupt their function, and a return of field of vision would not be at all unnatural, most particularly in the cases of encapsulated tumors, removed without damage to the surrounding brain.

7. So-called paradoxic diplopia following operations for strabismus, demonstrating that under certain circumstances binocular vision can have been present without association of fibers from corresponding retinal points.

8. The anatomic data of Adolph Meyer and T. J. Putnam, which demonstrated the widespread dispersion of the optic radiation, especially in its anterior portion.

SUMMARY

Asymmetry of incomplete homonymous hemianopic defects in the visual fields is a clinical entity regularly present in cases of lesions of the temporal lobe and decreasing in amount the farther back the lesion in the visual pathway, until in occipital lesions the congruity of the field defects becomes absolute, extending even to minute irregularities. This constant finding is only possible by means of careful quantitative perimetric study but is of great value in localizing cerebral lesion not only laterally but dorsoventrally.

The cause of these characteristic defects in the visual fields is to be found, it is believed, in the dissociation in the temporal lobe of homologous fibers from corresponding retinal points and their gradual coalescence in the postparietal area.

384 Post Street.

SYNDROME OF TUBEROUS SCLEROSIS

REPORT OF A CASE

FERDINAND L. P. KOCH, M.D.
Fellow in Ophthalmology, the Mayo Foundation

AND

MAURICE N. WALSH, M.D.*
ROCHESTER, MINN.

Since Bourneville[1] first described tuberous sclerosis of the brain in 1880 and van der Hoeve[2] in 1921 published the first known observations on the associated retinal tumors, other authors have reported additional cases of this rare disease. The condition is characterized by (1) retarded mental development, (2) the occurrence of epileptiform seizures and (3) the occurrence of tumors of one or more organs, usually the skin, the brain and the eye and often the liver, kidneys and heart and possibly the stomach. Variations from the typical clinical picture are not infrequent, as has been noted by Critchley and Earl.[3] They stated that only one component of the triad may be found. Instances have been reported in which only certain members of a family (in some cases, only one member) had tuberous sclerosis, whereas other members of the same family exhibited only one or two of the components of the typical triad of symptoms. Symptoms characteristic of tumor of the brain rarely occur in association with this entity without any clue as to the presence of tuberous sclerosis. In a few cases, however, only intra-abdominal lesions were present.

REPORT OF CASE

A youth aged 17, of English and Norwegian descent, was registered at the Mayo Clinic on Oct. 15, 1937. He complained of poor vision. The family history was irrelevant so far as was known. Rashlike, reddish, symmetrically distributed lesions of the skin about the face, neck and back had been noted at birth. The mother and sister of the patient believed that he had always been somewhat retarded mentally, but the relatives were unable to state at what age this initially was noted. Typical petit mal seizures appeared at the age of 2 months. These

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* From the Section of Neurology, the Mayo Clinic.

1. Bourneville, D.: Sclérase tubéreuse des circonvolutions cérébrales; idiotie et épilepsie hémiplégique, Arch. de neurol. **1**:81, 1880.

2. van der Hoeve, J.: Augengeschwülste bei der tuberösen Hirnsklerose (Bourneville), Arch. f. Ophth. **105**:880-898, 1921.

3. Critchley, M., and Earl, C. J. C.: Tuberose Sclerosis and Allied Conditions, Brain **55**:311-346 (Sept.) 1932.

increased in frequency until the patient reached the age of 7 months, when the seizures took place from four to six times daily, after which they decreased gradually until, at the age of 5 years, they had ceased entirely. Further attacks have not occurred.

Headaches first became manifest about three years prior to examination at the clinic. These were stated to be generalized and dull in character and increased gradually in severity. One year after their onset, the headaches appeared almost daily until, about two months prior to the patient's registration, they no longer occurred. The patient's only episode of vomiting took place about two weeks after cessation of the headaches. Progressive decrease in vision, which was slightly more extensive in the left eye than in the right, was first noted in April 1937. This manifested itself by the recurrent and sudden appearance of great blotchy white patches, which immediately obscured all but the largest of objects. The patient was forced to give up riding a bicycle but was able to get about on foot without assistance. The vision continued to decrease during the succeeding four months, and at the end of this period the visual acuity had become so poor that he could not walk outside the house unaided. At no time had there been intraocular or orbital pain. The course of events otherwise was irrelevant.

On general examination the patient was found to be well developed and well nourished. Roentgenographic examination of the thorax revealed an old healed lesion of osteomyelitis of the seventh rib on the left side. The stomach, heart and kidneys were examined roentgenographically, but without significant findings. Urinalysis revealed nothing remarkable. The formed elements of the blood were normal, and results of flocculation tests of the blood for syphilis were negative. The fats in the blood were studied carefully, but normal values were found for cholesterol esters, cholesterol, lecithin, fatty acids and total acids. Values for calcium and phosphorus in the serum were within normal limits.

Dermatologic examination revealed the symmetric distribution over the forehead, cheeks, chin and nasolabial folds of many small yellowish brown and reddish papillomatous tumors, which, on biopsy by Dr. Henry Brunsting of the Section on Dermatology and Syphilology, exhibited changes typical of adenoma sebaceum. Cutaneous papillomatous tags were found along the sides of the neck and in the axillary folds. Plaques of a deep reddish color, possessing little resistance on palpation, were noted on the skin of the midlumbar region. Café au lait areas were not observed (fig. 1).

The results of neurologic examination were objectively negative. Roentgenographic examination of the head revealed evidence of increased intracranial pressure with secondary erosion of the sella turcica, the tuberculum sellae and the wings of the sphenoid bone. Irregular rarefied areas indicative of calcification extending on both sides of the midline were found in the suprasellar region (fig. 2).

On ophthalmologic examination the vision of the right eye was found to have been reduced to the ability to count fingers at a distance of 2 feet (61 cm.) and of the left eye, to minimal perception of light. External ocular examination gave essentially negative results except for the presence of slight rotatory, bilateral nystagmus. The pupils were round and large and equal in size and became dilated regularly under the influence of mydriatics. The pupillary consensual light reflex occurred to a moderate degree and was bilaterally equal but was poorly sustained on the left side. The effect of pupillary constriction on accommodation was equal on both sides and was moderately active, but it was only moderately sustained. The right pupil contracted normally to direct light, and the left pupil contracted only perceptibly; constriction was only minimally sustained on the right and not at all on the left. Examination of the visual field of the right

eye elicited a nasal hemianopia with macular sparing, an enlarged blindspot and an altitudinal inferior anopsia. The visual field of the left eye was not obtainable. Responses were greatly delayed, and cooperation necessarily was poor. The localizing value of the visual field was negligible.

Roentgenographic examination of the optic foramens was attempted on two occasions, but cooperation was lacking, and the roentgenograms were unsatisfac-

Fig. 1.—Symmetrically distributed lesions of adenoma sebaceum.

tory; so far as could be determined, the roentgenograms would have been interpreted as normal.

The media were clear on ophthalmoscopic examination, and the vasculature was normal in appearance except for slight fulness of the veins together with smooth retinal arterioles, which showed mild generalized narrowing. The normal foveal reflex was not observed in either eye, and the maculas presented the appearance of central macular degeneration. Each nerve head was elevated fully 3 diopters, and there was pallor of each optic nerve of a moderately severe grade. These findings in association with old absorbing exudates and hemorrhages and

numerous fine new collateral capillary vessels were characteristic of receding chronic choked disks. Loss of nerve substance was not visible.

In the right eye there were two small nodular yellowish white tumors situated posteriorly, one being seen at a point 2 disk diameters below and temporal to the inferior margin of the optic nerve and 1.5 disk diameters below and nasal to the macula. The other mass was situated 4 disk diameters below and temporal to the macula. The appearance of these two lesions was strikingly similar. Each was elevated less than 1 diopter and was flat except for the yellowish white cyst-like nodules on its surface. These nodules were about eight in number and appeared as hemispheres placed against a highly refractile, white and slightly uneven base. The tumors were well demarcated and were irregularly circular, with shallow dentate margins. The retinal edema present as a result of the accompanying choked disks did not interfere with the apparent sharpness with

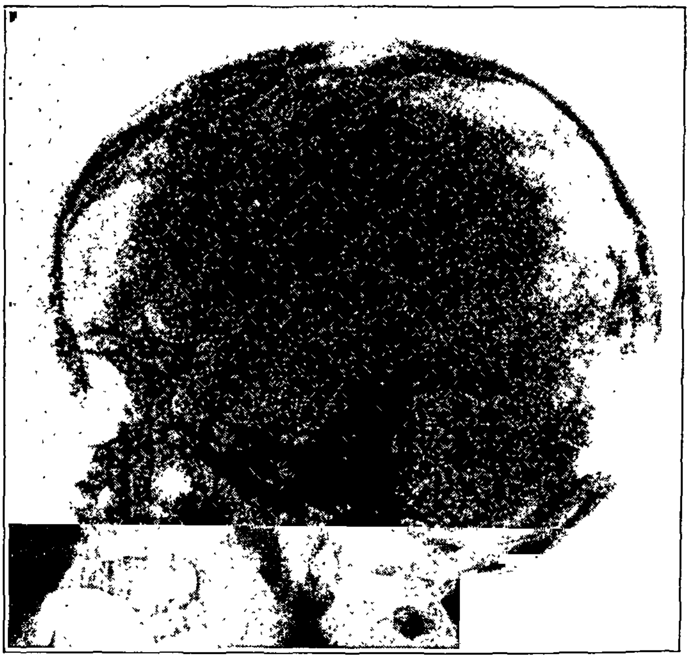

Fig. 2.—Roentgenogram of the skull. Just above the sella and extending on each side of the midline there is a large rarefied region indicative of calcification. Secondary to the increased intracranial pressure there is considerable destruction of the sella with erosion of the wings of the sphenoid bones and of the tuberculum sellae.

which the masses were defined ophthalmoscopically, but delineation of detail was somewhat reduced in the photographs of the fundi because of the nystagmus present. The avascularity of these tumors was striking and is in accord with the findings of others; however, both of these masses were to be seen in rather close physical association with secondary branches of the inferior temporal vein in that these vessels traversed their normal paths without deviation immediately adjacent to the edge or border of the tumors.

The ophthalmoscopic findings in both eyes were essentially identical except for the fact that only one mass was observed in the left fundus. This was situated far above the disk, slightly nasal to it and immediately beyond the midperiphery. The tumor measured slightly less than 2 disk diameters in length and 0.5 of a

disk diameter in width. The long axis of the tumor was parallel to the radius of the eye, taking the disk as the center of this circle. The inferior third of this tumor was elevated almost 1 diopter, and its surface presented a great many small, golden-yellow, cystlike granules against the white refractile surface of the tumor itself. This lower third was slightly greater in width than the upper two thirds of the mass and served to give the mass a rough club-shaped outline. The margins were well defined; blurring due to retinal edema was not present because the edema did not extend that far peripherally. The photograph of the fundus, however, depicted this demarcation and the superficial details only hazily because the tumor was too near the periphery to avoid distortion by the intervening ocular media. This tumor also was avascular but was in close proximity to the superior nasal vein, which appeared to traverse the base of the tumor without deviation when seen again on its emergence from the mass.

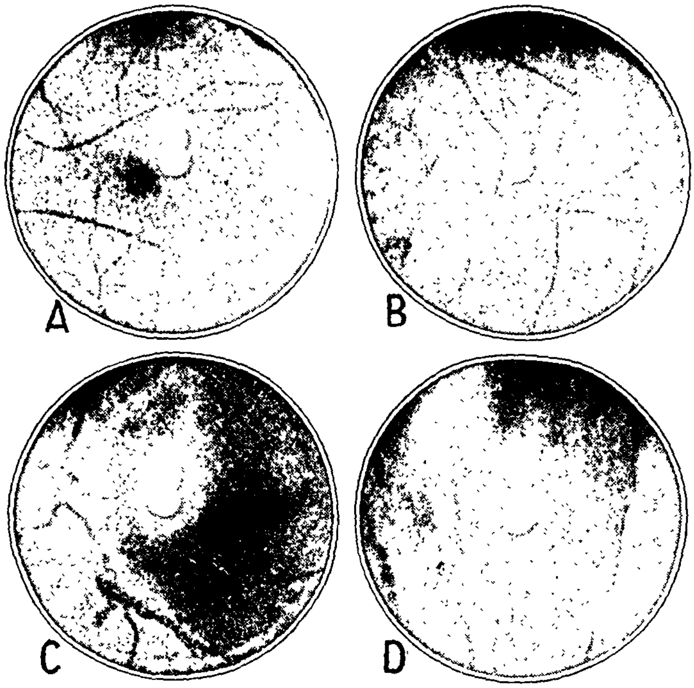

Fig. 3.—*A,* right fundus. One of the two tumors is shown. It is situated below the optic disk and the macula. Note the numerous small beadlike tumefactions. *B,* right fundus. The other of the two tumors, situated below the tumor shown in *A,* is evident. Note the beadlike tumefactions. *C,* left fundus. The region of the optic disk and macula, which was similar in appearance to the same region in the right eye, is shown. The small tumefactions are also present. *D,* left fundus. Delineation of the details of this tumor is not clear because of its situation so far out in the midperiphery above the disk that angular distortion by the ocular media could not be avoided. A few of the small tumefactions can be seen.

In addition to these relatively large tumefactions, there were many small punctate spots distributed over the retinal surface throughout the posterior fourth of each fundus. These smaller lesions were identical in appearance with the larger ones except for the difference in size, and it was thought that they represented manifestations of the same process. Many of these punctate spots were strung along the veins like beads, and a few were noted in the immediate vicinity of the macular regions. Tumors of the optic disk were not present (fig. 3).

The appearance of the eyegrounds remained relatively unchanged during the eighteen days that the patient was under observation. It had been observed by van der Hoeve[4] that the nodulations of the surfaces of these tumors are cysts which undergo an almost constant process of rupturing, emptying and refilling. He stated that he had seen one of these cysts drop off into the vitreous after constriction of the neck of the individual cyst had occurred, and later, on examination, he noted that more tumors were present than at the previous examination. Thus he felt that he had observed the actual process of metastasis as it occurred by intraocular implantation.

In our case the number of these nodulations or cysts seen varied slightly from time to time or were less prominent at one examination than at another; however, the media remained equally clear at all times and under the same examining conditions. The only progressive change noted was the bilaterally diminishing papilledema from that of 3 diopters to 2 diopters, accompanied by a slight relative increase in the pallor of the optic nerves but without demonstrable loss of nerve substance. It was not established that these changes were associated with high voltage roentgen therapy that the patient had been receiving.

At the time of the patient's dismissal from observation, visual acuity had been reduced to the ability to count fingers at a distance of 1 foot (30 cm.) with the right eye and to the most minimal degree of perception of light with the left eye.

The mother and sister of the patient were examined ophthalmoscopically, but with negative results. None of the stigmas of tuberous sclerosis were observed.

A diagnosis of tuberous sclerosis was made on the basis of the history, the presence of the associated lesions of adenoma sebaceum, the typical retinal tumors and a large calcified tumor probably invading the third ventricle with associated increase of intracranial pressure. Dr. A. W. Adson saw the patient in neurosurgical consultation and advised that operation should not be performed because of the patient's relatively poor condition and because of the inaccessibility of the tumor. Accordingly, high voltage roentgen therapy was administered to the head, and the patient was dismissed from the clinic on Nov. 3, 1937. So far as is known, there was little change in his general condition when information was last obtained nine months later, in August 1938.

COMMENT

The etiology of tuberous sclerosis is unknown, but the condition probably is hereditary in origin. It may represent, in some instances at least, a recessive characteristic. Fabing[5] reported its occurrence in identical twins, and it has also been reported as occurring in members of three generations of one family by Kirpicznik[6] as well as in five of nine siblings by van Bouwdijk Bastiaanse and Landsteiner.[7] On the

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4. van der Hoeve, J.: Eye Symptoms in Phakomatoses, Tr. Ophth. Soc. U. Kingdom **52**:380-401, 1932.

5. Fabing, H.: Tuberous Sclerosis with Epilepsy (Epiloia) in Identical Twins, Brain **57**:227-238 (Oct.) 1934.

6. Kirpicznik, J.: Ein Fall von tuberöser Sklerose und gleichzeitigen multiplen Nierengeschwülsten, Virchows Arch. f. path. Anat. **202**:358-376 (Dec. 3) 1910.

7. van Bouwdijk Bastiaanse, F. S., and Landsteiner, K.: Eine familiäre Form tuberöser Sklerose, abstracted, Zentralbl. f. d. ges. Neurol. u. Psychiat. **32**:197, 1923.

contrary, as in our case, the family history may be entirely irrelevant. Sherlock [8] termed the syndrome epiloia, and this name occasionally appears in the literature. Brouwer, van der Hoeve and Mahoney [9] in a recent report pointed out under the generic term of phakomatosis the apparently remarkable similarity between tuberous sclerosis, Recklinghausen's neurofibromatosis, von Hippel-Lindau's angiomatosis and the entity of the Sturge-Weber synlrome.

Pathologic studies were made only of the skin in our case; however, in cases in which more extensive studies were possible, evidence of widespread alteration of tissue has been found. The lesions in the brain appear typically in regions of the cerebral cortex. These are firm to the touch and gave rise originally to the name tuberous sclerosis, or potato-like (tuberose) sclerosis. In these regions the normal lamination of the cortex is disturbed, and the cytoarchitecture is disorganized. These tumors contain both glial and neuronal elements. Gliosis and, in many cases, demyelination may occur in the white matter. Multiple mixed types of tumors of the brain characteristically occur. These are found most frequently near the lateral ventricles, into which they may protrude. Their appearance on gross section has been described as closely resembling that of candle drippings.

Rarely, as in our case, the clinical picture of increased intracranial pressure may be produced by the presence of one or more large intraventricular tumors. These often are atypical spongioblastomas. There may occur, in addition to the tumors of the skin which usually are present, mixed tumors of the liver and kidneys, rhabdomyomas of the heart, myomas of the gastrointestinal tract and retinal tumors. The renal tumors are said to be identical with the Wilms tumor.

Several hypotheses have been advanced to explain the origin of these interesting lesions of the brain. It was thought at one time that they represented a manifestation of syphilis, but this is now known to be incorrect. Many authors still hold the early view that the responsible factor is a developmental disorder occurring in embryonic life. Bielschowsky [10] expressed the belief that the process possibly is both a developmental malformation and an additional neoformation. Ferraro and Doolittle [11] recently suggested the name diffuse neurospongioblastosis

8. Sherlock, E. B.: The Feeble-Minded: A Guide to Study and Practice, London, Macmillan and Company, 1911.

9. Brouwer, B.; van der Hoeve, J., and Mahoney, W.: A Fourth Type of Phakomatosis: *Sturge-Weber Syndrome, K. Akad. v. Wetensch. te Versl. Amsterdam* (Sect. 2) **36**:1-33, 1937.

10. Bielschowsky, M.: Zur Histopathologie und Pathogenese der tuberösen Sklerose. Neue Beiträge, J. f. Psychol. u. Neurol. **30**:167-200, 1924.

11. Ferraro, A., and Doolittle, G. J.: Tuberous Sclerosis (Diffuse Neurospongioblastosis), Psychiatric Quart. **10**:365-416 (July) 1936.

on the premise that the process is neoplastic. Lipoid deposits in the brain have been observed in some cases, and this has led to the suggestion that the disease might be the result of an embryologic disorder of metabolism of lipoids. It is of interest to reiterate here that in our case the values for lipoids in the blood were well within normal limits.

Tumors of the retina in cases of tuberous sclerosis were first noted in 1921 by van der Hoeve,[2] who reported a series of 6 cases. There have since appeared in the literature at least 24 additional authentic cases, and of these, tumors of the retina proper have been the more common, there having been reported only 6 situated on or very near the optic disk. A seventh, Walsh's case of a tumor situated on the disk, does not appear in the reports, but a photograph of the retinal pathologic process is recorded.[12] Messinger and Clarke,[13] Hopwood,[14] Kveim[15] and Bloch and Grove[16] in recent papers included bibliographies on this subject.

The rarity of this retinal pathologic entity militates against its more frequent recognition on ophthalmoscopic examination. Undoubtedly the lesion has occurred much more often than is indicated by the number of reports in the literature. The present ratio is approximately 1 to 5, but in all probability this would have been more nearly equalized had the ocular fundi been examined carefully in each instance.

Tumors which are situated at the disk are passed over less easily because of their greater size, their elevation forward into the vitreous and the whitish, coarsely nodular or cystic, refractile surfaces. The smaller tumors, of which there may be one or more, are found usually in the posterior third of the fundus.

Gottlieb and Lavine[17] mentioned a solitary, partially vascularized retinal tumor in their case. Van der Hoeve[18] briefly mentioned finding

12. Ford, F. R.: Diseases of the Nervous System in Infancy, Childhood and Adolescence, Springfield, Ill., Charles C. Thomas, Publisher, 1937, p. 748.

13. Messinger, H. C., and Clarke, B. E.: Retinal Tumors in Tuberous Sclerosis: Review of the Literature and Report of a Case, with Special Attention to Microscopic Structure, Arch. Ophth. **18**:1-11 (July) 1937.

14. Hopwood, A. T.: Tuberous Sclerosis: Report of Five Cases Including One Case in One of Twins, Ohio State M. J. **33**:277-282 (March) 1937.

15. Kveim, A.: Ueber Adenoma sebaceum (Morbus Pringle), und seinen Platz im neurokutanen Syndrom—tuberöse Gehirnsklerose—und dessen Beziehung zur v. Recklinghausenschen Krankheit: Drei Fälle von tuberöser Sklerose und Morbus Pringle mit röntgenologisch nachweisbaren Veränderungen in Cerebrum und im Knochensystem, und Tumorbildungen in Retina, Acta dermat.-venereol. **18**:637-683 (Oct.) 1937.

16. Bloch, F. J., and Grove, B. A.: Tuberous Sclerosis with Retinal Tumor: Report of a Case, Arch. Ophth. **19**:34-38 (Jan.) 1938.

17. Gottlieb, J. S., and Lavine, G. R.: Tuberous Sclerosis with Unusual Lesions of the Bones, Arch. Neurol. & Psychiat. **33**:379-387 (Feb.) 1935.

18. van der Hoeve, J.: Eye Diseases in Tuberose Sclerosis of the Brain and in Recklinghausen's Disease, Tr. Ophth. Soc. U. Kingdom **43**:534-541, 1923.

a single "typical" tumor of tuberous sclerosis in the left eye of a woman aged 21, without other signs or symptoms but in whose family five siblings were found subsequently to be afflicted with advanced forms of the disease. This patient was seen six years later, during which time she had married.[9] The retinal tumor had become somewhat enlarged, and a few smaller ones had appeared. Retinal tumors were found in three of her six children, and a fourth had numerous epileptic attacks. It would appear, therefore, that the finding of nothing more than one of the typical retinal tumors justifies a positive diagnosis of tuberous sclerosis.

These tumors appear to originate from the nerve fiber layer of the retina and are rather flat. Elevation, if measurable, is usually less than 1 diopter. The growth is seldom greater than 1 disk diameter in any axis and is always oval or circular, with well defined but slightly dentate margins. It may be yellowish white or yellowish gray and is in sharp contrast with the normal red fundus reflex, a contrast that is heightened by the refractile quality of the surface. Fine to relatively coarse cyst-like nodules invariably have been noted on the surface of these masses and have been likened to the external appearance of an unripe mulberry.[19] Few observers have failed to comment on the apparent avascularity of these tumors, and although some cases have been reported in which the tumors possessed a well defined blood supply, the majority of growths have been described as existing in only a casual physical association with any retinal vessels. The retinal vasculature always has been reported as normal except in those few instances in which secondary local changes in the vasculature have occurred subsequent to choking of the disks in conjunction with the intracranial lesions. Tumors occupying the immediate macular region have not been reported.

On casual observation, particularly if the refractive media are hazy, one may interpret the tumefactions as areas of gliosis or of choroidal degeneration. The tumors, however, are confused more easily with von Hippel-Lindau's angiomatosis of the retina and with the neurofibromatosis of Recklinghausen's disease. In cases of angiomatosis, even in the early stages of the process, the intimate relationship of the small angioid tumor with the retinal vessels presenting the typical changes will serve to distinguish the one from the other; in cases of neurofibromatosis the tumors are also small and rather well vascularized and are usually flat and not numerous. In addition, of course, the diagnosis is determined by the history and by the results of general and neurologic examinations.

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19. Vogt, A.: Seltener Maulbeertumor der Retina bei tuberöser Hirnsklerose, 9 Jahre verfolgt, Ztschr. f. Augenh. **84**:18 (Aug.) 1934.

So far as can be determined from the literature, visual disturbances in cases of tuberous sclerosis are limited to a subjective decrease in vision, which can be confirmed objectively. The loss of vision, however, is not due to the presence of tumefactions in the retina, there having been none reported as being situated in the immediate region of the macula, but it does occur subsequent to formation of a massive tumor at the papilla or to prolonged increased intracranial pressure with chronic choking of the disks and atrophy of the optic nerve. One such case has been reported by Guillain and Lagrange.[20]

Retinal tumors of this type have been classified with the accompanying visceral tumors,[21] but this, of course, is erroneous if the embryologic origin of the eye is considered.[22] The disease entity, tuberous sclerosis, appears to be the result of a developmental anomaly commencing early in fetal life and eventuating in widespread metaplasia, which affects chiefly the ectodermal tissues but with subsequent prevalence of both mesodermal and entodermal derivatives.[23] Thus, when the typical intracranial lesions are known to be present, it would be reasonable to predicate the demonstration of the characteristic retinal pathologic process.

As Grinker [24] pointed out, the classification of these tumors remains uncertain, there having been an insufficient number reported, and too few of such lesions have been subjected to microscopic examination. It is of interest to mention here an isolated tumor of the left eye recently reported by McLean [25] as an astrocytoma, or true glioma of the retina, because ophthalmoscopically it was not unlike the retinal tumors in cases of tuberous sclerosis reported by some observers.

Tumors of the optic papilla have been studied microscopically in 2 cases, in 1 by van der Hoeve [4] and in 1 by Messinger and Clarke,[13] and those of the retina in 4 cases, in 1 each by van der Hoeve,[4] Schob,[26]

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20. Guillain, G., and Lagrange, H.: Phacomatose rétinienne de Van der Hoeve dans un cas de sclérose tubéreuse, Bull. et mém. Soc. méd. d. hôp. de Paris **50:** 1421-1425 (Nov. 12) 1934.

21. Critchley and Earl.[3] Ferraro and Doolittle.[11]

22. Mann, I. C.: Development of the Human Eye, London, Cambridge University Press, 1928.

23. Yakovlev, P. I., and Guthrie, R. H.: Congenital Ectodermoses (Neurocutaneous Syndromes) in Epileptic Patients, Arch. Neurol. & Psychiat. **26:**1145-1194 (Dec.) 1931. Critchley and Earl.[3]

24. Grinker, R. R., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1058.

25. McLean, J. M.: Astrocytoma (True Glioma) of the Retina: Report of a Case, Arch. Ophth. **18:**255-262 (Aug.) 1937.

26. Schob, F.: Beitrag zur Kenntnis der Netzhauttumoren bei tuberöser Sklerose, Ztschr. f. d. ges. Neurol. u. Psychiat. **95:**731-740. 1925.

Feriz [27] and Kuchenmeister,[28] respectively. The methods of preparation and the technics of staining, however, appear not to have been sufficiently adequate for a definite determination of the type of cell contained in these tumors.

Van der Hoeve [4] saw no essential histologic difference between the retinal tumors and those of the papilla. He expressed the belief that the typical cells were so-called glioneurocytes which had not yet become differentiated into glial cells or ganglion cells and that they were descendants of the first anlage of the retina. According to him, the tumor originated in the layer of nerve fibers. Messinger and Clarke [13] in 1937 reported their observations on histologic preparations of a solitary tumor obtained in a case in which death had occurred due to tuberous sclerosis. Greenfield [29] demonstrated for these authors short fine fibrils in the processes of the larger cells by means of phosphotungstic acid. Mann [30] examined sections of the specimen and expressed the belief that the cells were glial in origin, probably having been derived from glial cells of the inner neuroblastic layer during the second stage of retinal differentiation, which takes place from the sixth week to the third month of intrauterine development.

Messinger and Clarke [13] suggested that these retinal tumors might be considered as hamartomas rather than as true tumors. Grinker [23] stated that they probably are astroblastic in origin and are of slow growth and that the characteristic cell is the same as that found in the nodules in the brain. The cells of the retinal tumors are large and contain much cytoplasm and present many fine glial processes which arise from the body of the cell. The arrangement is always syncytial, and mitotic figures are found infrequently. Growth of the tumor in the vitreous and in the lens was described by van der Hoeve,[18] who considered this evidence of malignancy. Cytologically, however, together with the tumor reported by McLean,[25] these growths appear to be relatively benign. By analogy, they should be of a low degree of malignancy if they are astroblastic in origin, because astrocytomas of the brain are the least malignant of neural tumors.

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27. Feriz, H.: Ein Beitrag zur Histopathologie der tuberösen Sklerose, Virchows Arch. f. path. Anat. **278**:690-769 (Oct. 4) 1930.

28. Kuchenmeister, E.: Ueber einen Fall von Pringlescher Krankheit mit Veränderungen am Augenhintergrund und an den Schleimhäuten von Blase und Mastdarm, Dermat. Wchnschr. **99**:1333-1337 (Oct. 13) 1934.

29. Greenfield, J. G., cited by Messinger and Clarke.[13]

30. Mann, I., cited by Messinger and Clarke.[13]

OPERATIVE TREATMENT OF RADIATION CATARACT

ALGERNON B. REESE, M.D.
NEW YORK

At the Memorial Hospital for the Treatment of Cancer and Allied Diseases, I had the opportunity to examine 112 eyes with radiation cataract and to operate on 25 of these at the Institute of Ophthalmology. Certain conclusions have been reached regarding the operative treatment in these cases, and it is this phase of the subject that will be presented in this paper. The clinical characteristics, the histologic structure and the other aspects of the subject will be given only as they are related to the operative treatment. In this report an effort has been made to include only cataracts that have unmistakably resulted from irradiation. The criteria have been as follows: A sufficiently large dose of radiation reaching the eye, the appearance of cataractous changes after the usual latent period of from two to three years, the location of cataractous changes in a characteristic manner in the posterior subcapsular region and the manner of development into maturity. Slit lamp examination in all of the cases in which operation was performed revealed an important finding. An anterior subcapsular haze was seen after the posterior subcapsular opacity had developed. This haze was usually composed of fine striations and became increasingly more dense, particularly in the central portion where in some instances it ultimately produced a well demarcated white area. These subcapsular changes were due to a proliferation of the lens epithelium, which will be discussed more fully later.

Of the 25 eyes operated on, intracapsular extraction was done on 16, extracapsular extraction on 6 and linear extraction on 3.

INTRACAPSULAR EXTRACTIONS

Technic.—For intracapsular extraction, the usual incision with a conjunctival flap is made. This is enlarged a little with Steven's scissors on each side, so that it extends over 180 degrees of the limbic circumference. A small iridectomy is performed. A double-armed atraumatic black silk suture 6 O is placed so that it

From the Institute of Ophthalmology and the Memorial Hospital for the Treatment of Cancer and Allied Diseases.

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includes limbic tissue on the one side and a substantial amount of the conjunctiva and its underlying episcleral tissue on the other side. With the Arruga forceps, a rather wide bite of the anterior lens capsule is obtained over the lower one-third. Counterpressure is exerted with the strabismus hook over the sclera near the limbus below, and the zonules are ruptured over the lower part by gentle manipulation of the lens with the forceps. Pressure with the hook is partially abandoned, and the dislocated lower part of the lens is pulled forward and upward until it appears in the wound. The lens is then lifted straight out, having been half tumbled. The suture is tied, and the iris pillars are replaced. A 3 per cent solution of atropine sulfate is instilled, and a dressing is applied to both eyes.

Results.—Operation by the intracapsular method was done consecutively on 16 eyes. The capsule did not tear in any case, and the zonules ruptured about as readily as usual. Vitreous was lost in 1 instance. This was the sixteenth operation of the series, and the vitreous appeared after the incision was made and before the lens was grasped with the capsule forceps. The corrected vision was ultimately 20/15, and the patient could read Jaeger test type 1. The corrected vision in 9 of the other cases was 20/20 or better; in 6 it was 20/40 or better and in 1, 20/100. There were slight senile degenerative changes in the macula in the last case. There were no instances of secondary glaucoma after the operation. In 1 case secondary glaucoma developed the fourth month after operation from occlusion of the central retinal vein. In only 1 case was postoperative hemorrhage noted, and in this case hyphema, measuring 3 mm. in diameter, occured on the seventh day after the operation, but absorbed readily.

HISTOLOGIC PICTURE

The 16 lenses removed in capsule were examined microscopically, and the histologic picture relevant to the operative treatment in these cases is given here.

Under the anterior capsule the epithelial layer showed various signs of proliferation. Instead of having a thickness of a single cell, it varied from several to innumerable cells (fig. 1, *a*). These showed all metaplastic transitions, from the normal-appearing epithelial type to the long connective tissue-like cells with a fibrous intercellular structure. Under the capsule there occurred in some instances zones of cortical liquefaction (fig. 1, *b*), which were rather sharply outlined from the surrounding lens substance. At the deeper margins of these areas of softening the epithelial nuclei were sometimes discernible (fig. 1, *c*). The epithelium in places produced rudimentary and malformed lens fibers under the capsule. These appeared as sharply demarcated, vesicle-like cells, some of which showed nuclei and some of which did not (fig. 2, *a*). In places the subcapsular epithelium was missing

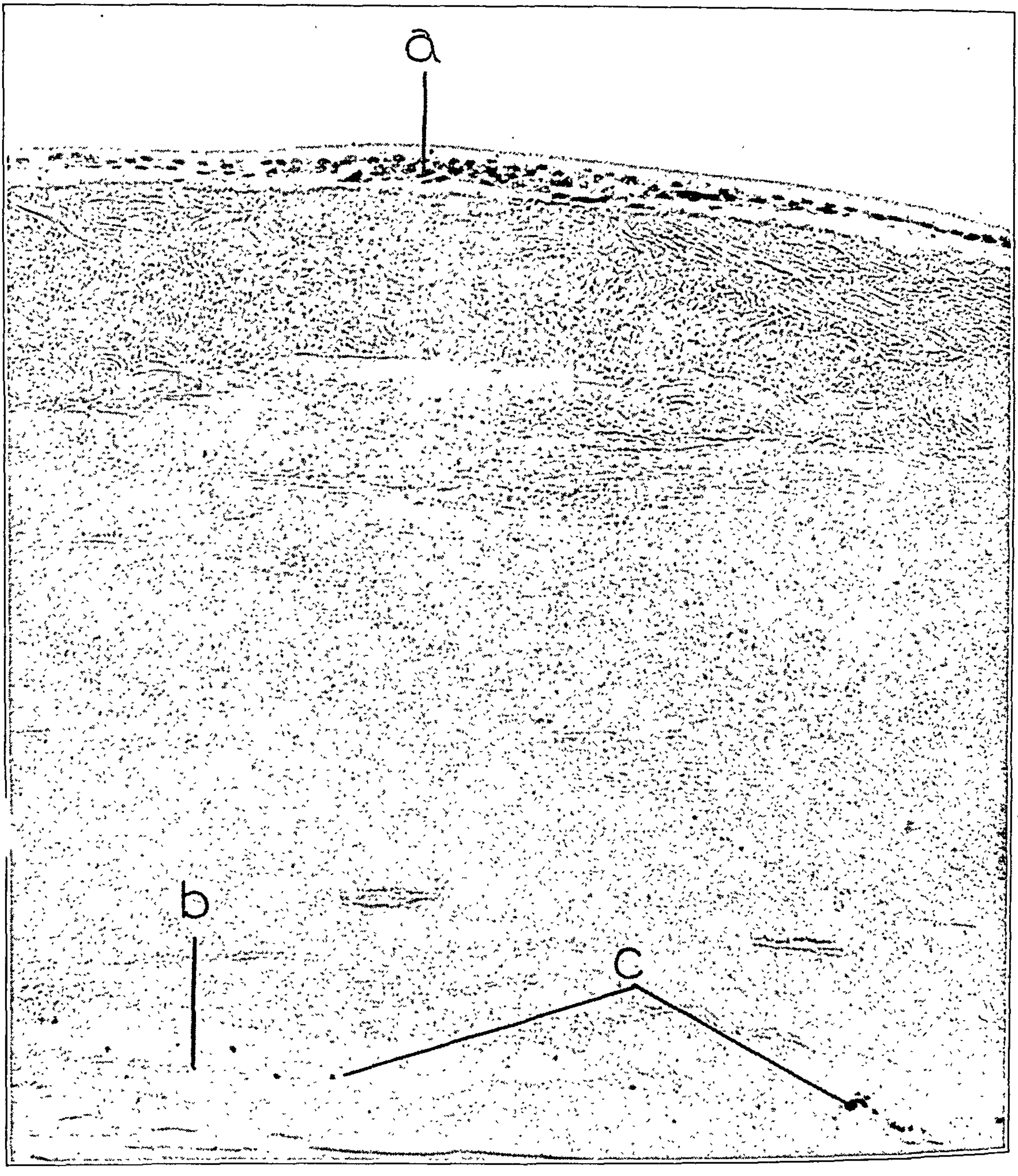

Fig. 1.—Under the anterior capsule at *a* the epithelium has proliferated and undergone some metaplastic changes. The cortex as far as *b* is liquefied. Some proliferated epithelial cells are seen at the margin of the liquefaction at *c*.

entirely. In fully matured cataracts of longer duration the proliferated epithelium showed a more complete metaplastic change into fibrous-like tissue under the anterior capsule; it was thickest in the central portion and gradually thinned toward the periphery (fig. 3, *a*). This fibrous plaque of tissue under the anterior capsule was quite thick and dense in some instances, and its contracture produced folds in the overlying capsule. The epithelial cells tended to proliferate and extend also under the posterior capsule. Sometimes they were normal-looking cells, but

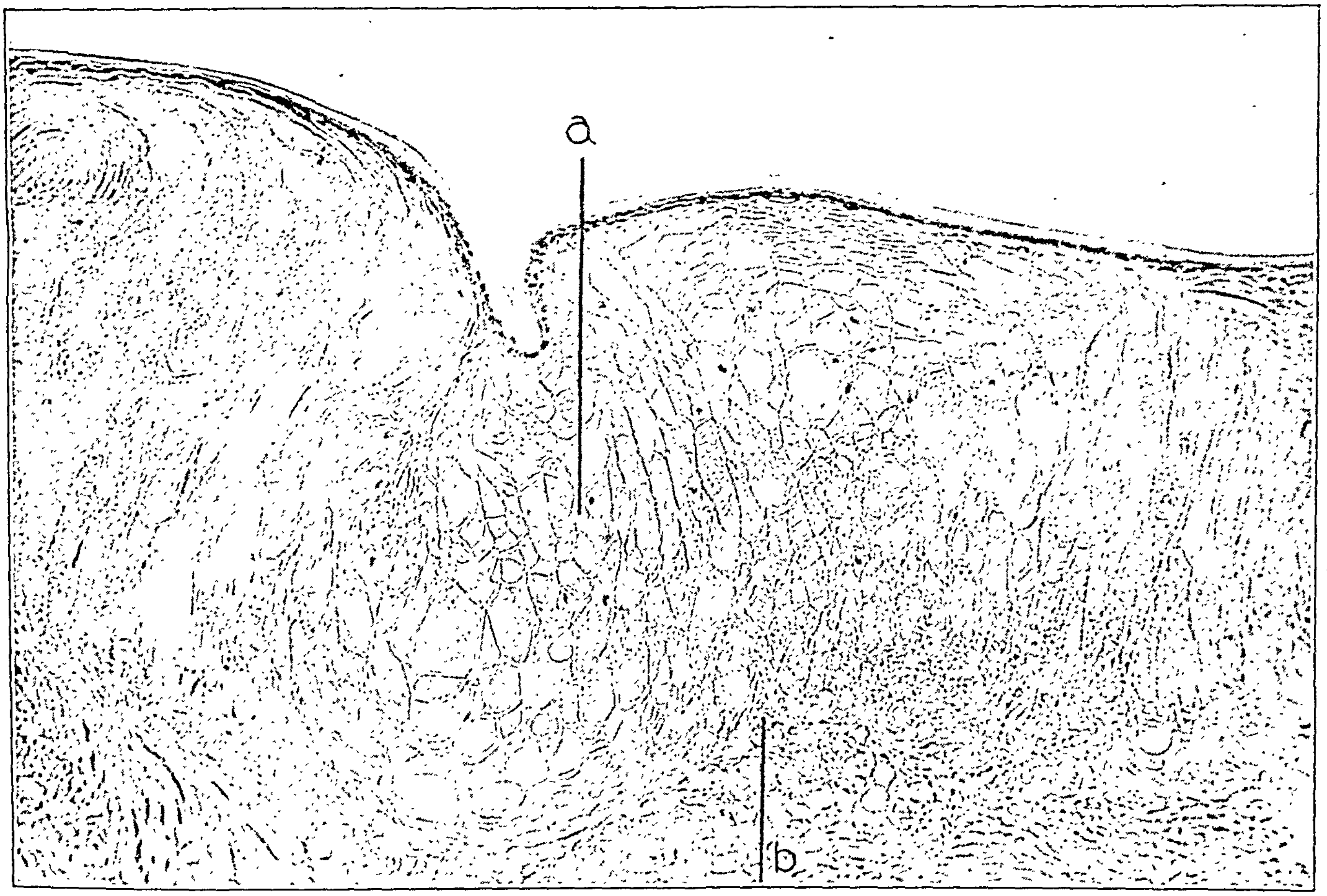

Fig. 2.—Under the anterior capsule the epithelium has proliferated into several layers thick. A layer of malformed vesicle-like lens fibers (*a*) has been produced as far as *b*. The nuclei of some are shown.

more often they had a long nucleus, resembling connective tissue cells, and frequently these atypical epithelial cells tended to form rudimentary and malformed lens fibers. Foci of liquefaction were also seen under the posterior capsule, and atypical epithelial cells proliferated sometimes into or around it. The germinative area, or nuclear zone, at the equator of the lens sometimes showed more, and sometimes less, active proliferation than usual. Occasionally, the nuclei were almost absent, and the lens substance in this region was fragmented or liquefied.

Sometimes atypical vesicle-like lens fibers were formed by these equatorial cells. Proliferation of the lens epithelium from irradiation has been reported by Grzedzielski.[1]

Interpretation.—The subcapsular and equatorial epithelium may receive in part a lethal dose and in part a sublethal dose of radiation. As a result of the former, the epithelium disappears, and as a result of the latter, it is stimulated to proliferate. This proliferation is an active process which not only replaces the destroyed epithelium but continues with little or no abatement. The longer the duration of the process, the more the proliferated epithelial cells show the metaplastic change into the fibrous tissue and the denser and thicker this tissue becomes. This more advanced stage of proliferation and metaplasia is seen only under the anterior capsule. This older and denser fibrous tissue

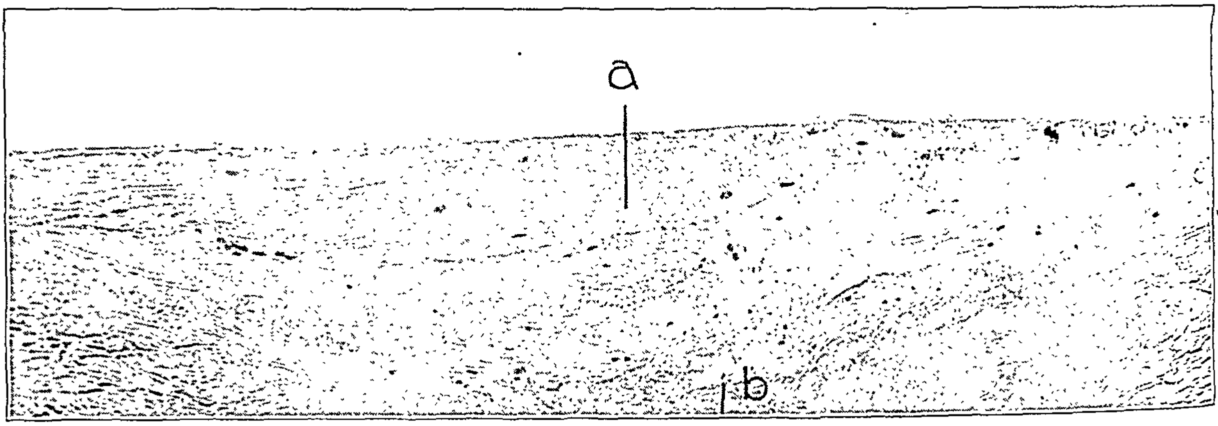

Fig. 3.—Under the anterior capsule is a layer of rather dense fibrous-like (*a*) tissue extending into the cortex as far as *b*. Many of the nuclei are seen.

(fig. 3, *a*) may be a condensation and inspissation of the malformed, vesicle-like fibers (fig. 2, *a*).

A LENS REMOVED IN CAPSULE WITH THE SHARP-TOOTHED FORCEPS OF TERSON

One of the cases in which an extracapsular extraction was to be performed warrants some details:

Before the operation it was noted that an area just under the anterior surface of the lens in the central part was more opaque than the surrounding region and was dull white. This area, which had irregular margins, faded out gradually toward the periphery of the lens. There were three small posterior synechiae at the pupillary margin below. The cornea showed some diffuse opacities and a few

1. Grzedzielski, J.: Zur Histologie der Röntgenkatarakt, Klin. Monatsbl. f. Augenh. **60**:95, 1935.

blood vessels which were produced by irradiation. Because of the synechiae, it was elected to do an extracapsular extraction. It was impossible to cut the anterior capsule with the cystotome except in the periphery, but an opening was obtained here, as evidenced by the escape of some milky fluid. The anterior capsule was then grasped with Terson's sharp-toothed forceps in an attempt to remove it. The capsule would not tear, so the lens was dislocated and removed in capsule with the sharp-toothed forceps. The operation was without incident, and the final corrected vision was 20/40. The reduced vision was due to the corneal opacity. This lens was sectioned and examined microscopically. Under the anterior capsule there was, instead of the epithelium, a layer of dense fibrous tissue six times thicker than the anterior capsule. This tissue was thickest in the central part and gradually thinned out toward the periphery, so that it almost disappeared in the region of the equator.

This tissue explains (1) the nature of the dense white area seen clinically under the anterior surface of the lens, (2) the reason it was so difficult to open the anterior capsule with the cystotome and (3) the reason the lens could be delivered in capsule with a sharp-toothed capsule forceps without tearing the capsule.

EXTRACAPSULAR EXTRACTIONS

Technic.—The technic for extracapsular extraction is the same as that for intracapsular extraction except that a large square area of anterior capsule is incised with the cystotome, and this piece of capsule is removed with the Terson forceps. The nucleus of the lens is then expressed, and usually any soft lens matter is irrigated from the anterior chamber with physiologic solution of sodium chloride.

Results.—In each of the 6 extracapsular extractions the nucleus of the lens delivered without difficulty and without loss of vitreous. Three of the patients had an uneventful convalescence, the ultimate vision of 2 being 20/30 and that of 1 being 20/20. A discission was necessary after three months on 1 of these patients; 1 of the other 2 died several months after operation, and the other could not be traced, so that it was not known whether a secondary membrane developed. The other 3 cases will warrant a more detailed report.

Case 1.—Before the operation, examination of the eye gave negative results except for the radiation cataract. The intraocular tension taken with the Schiötz tonometer was 17 mm. of mercury. A combined extracapsular extraction was done, and the operation was without incident. Postoperatively, the anterior chamber was shallow for ten days. Six weeks after operation the tension was 22 mm. of mercury. Seven weeks afterward the corrected vision was 20/20. Nine months postoperatively the tension was found elevated for the first time, measuring 34 mm. of mercury. There had been almost a continuous slight blush to the eye within the limbic region due to a mild iridocyclitis. The tension ranged between 34 and 45 mm. of mercury even with miotics, so that cyclodialysis was done two months before the time of writing, with the result that the tension has ranged from 9 to 11 mm. of mercury since. Several months after operation there appeared a

grayish white tissue in the coloboma and around the pupillary area. This became increasingly more dense but left a central clear area, which permits good vision.

CASE 2.—Preoperative examination of the eye gave negative results except for the radiation cataract. The tension was 20 mm. of mercury with the Schiötz tonometer. A combined extracapsular extraction was done, and the operation was without incident. For ten days the anterior chamber was shallow. The eye remained slightly congested due to a mild iridocyclitis, and two months after operation the tension was 29 mm. of mercury and a hemorrhage occurred in the anterior chamber, although there was no history of trauma. It required about two weeks for it to absorb. Five months after operation a mild iridocyclitis was still present, and a dense grayish white membrane filled the coloboma and the pupillary area. The intraocular tension was elevated to 35 mm. of mercury. This ranged from 35 to 45 mm. of mercury for the next month, so that cyclodialysis was necessary. The following three months the tension was found once as high as 40 mm., but most of the time it was normal. The grayish white tissue occupying the coloboma and the pupillary area had become so extremely dense that it was necessary to use de Wecker scissors to cut an opening in it. The tension fluctuated for the following five months, but usually it was elevated to around 35 mm. of mercury. A slight pannus with bullous keratitis formed, and vision of only 20/200 could be obtained.

CASE 3.—Before the operation there were noted some newly formed blood vessels in the bulbar conjunctiva and a few in the cornea, which were due to irradiation. A combined extracapsular extraction was done without incident. After the operation there were continued lacrimation, pain and conjunctival and ciliary congestion due to iridocyclitis and secondary glaucoma. The pupillary area and coloboma gradually became occluded by a dull, grayish white opaque tissue, which seemed to grow progressively more dense and vascularized. This tissue seemed to extend across the coloboma toward the site of the wound. The iris showed indistinct surface markings and many newly formed blood vessels, and around the margins of the pupillary area and the coloboma the iris was adherent to the newly formed tissue and merged gradually into it. After eight months the eye was enucleated because of pain.

Microscopic examination showed a thick, dense fibrous membrane filling the pupillary area and operative coloboma. This membrane extended from the posterior aspect of the operative wound at the limbus above to the posterior surface of the iris below. It was adherent to the iris and encompassed the pillars of the iris. It was intimately associated with the remains of the lens epithelium which had been left in the Soemmering ring. There was a narrow anterior peripheral synechia and an iris bombé. The stroma of the iris showed an eosin-staining fluid in the interstices and a considerable number of lymphocytes, which were also found to a less extent in the ciliary body. The anterior hyaloid membrane was intact. The retina, choroid and optic nerve were not remarkable.

The dense grayish white tissue seen clinically in the coloboma and the pupillary area represented a fibrous metaplasia of the proliferated lens epithelium. It seems probable that this was responsible for the iridocyclitis and secondary glaucoma.

Summary.—In 3 cases there was the usual postoperative course, and ultimately normal vision was obtained.

In 3 cases iridocyclitis and secondary glaucoma occurred after the operation. In each instance this was accompanied by the formation of a dense membrane in the pupillary area and the coloboma, varying from gray to pearl white. One of these eyes was enucleated because of pain. Microscopic examination of this eye, together with the clinical course of these cases, seemed to indicate that when an extracapsular extraction is done on a radiation cataract, iridocyclitis and secondary glaucoma tend to occur. The lens epithelium, left in the eye, has been stimulated by the irradiation to proliferate and continues to do so after the operation, thus producing a dense fibrous-like tissue which seems to be responsible for the iridocyclitis and secondary glaucoma.

LINEAR EXTRACTIONS

Technic.—For linear extraction, the pupil is dilated, and a Ziegler knife needle is passed into the anterior chamber at the limbus under a small conjunctival flap. The anterior capsule is opened by a Y incision. After from five to seven days a keratome incision is made in the cornea above, 3 mm. from the limbus. The soft lens matter is washed out with physiologic solution of sodium chloride.

Results.—Linear extraction was performed in 3 cases, 2 of which are reported here.

Case 1.—A boy 11 years of age had bilateral radiation cataracts. Linear extraction was done on the right eye, and in six weeks the corrected vision was 20/20. In two and a half years the vision had gradually grown worse, so that it was ultimately reduced to 20/200 because of a secondary membrane. A discission was done, which resulted in a corrected vision of 20/20. Linear extraction was done on the left eye, and in two months the corrected vision was 20/20. In one and a half years the vision had gradually grown worse, so that it was ultimately reduced to 20/100 because of a secondary membrane. A discission was done, which resulted in a corrected vision of 20/20.

Case 2.—A girl 6 years of age had had bilateral retinoblastoma. One eye had been enucleated and the fellow eye irradiated. A cataract developed which was a dense pearly gray and was located just under the anterior capsule. The density was greatest centrally and diminished toward the periphery. Discission was attempted, but the anterior capsule was so tough that an opening could not be made in it with a Ziegler knife-needle or a Wheeler discission knife. As the projection of light was questionable and the eye was soft, no further operative procedure was tried.

The dense opacity seen just under the anterior capsule was unquestionably fibrous tissue from proliferated lens epithelium, and the inability to open the lens was due to this.

PROLIFERATION OF LENS EPITHELIUM IN SENILE AND COMPLICATED CATARACTS

Proliferation of the lens epithelium is seen in some senile cataracts, and particularly those of long standing. This proliferated epithelium

may also undergo fibrous metaplasia and be seen clinically as an opaque, whitish area under the anterior capsule, densest in the central part of the lens and thinning toward the equator. I believe that this epithelial proliferation, even in the less marked instances, can be appreciated with the slit lamp as a subcapsular change. When this is present, the capsule should be sufficiently strong to withstand an intracapsular extraction. It may be that these changes, as well as the frequently mentioned weakened zonules, make lenses in elderly persons suitable for intracapsular extraction.

Proliferation of the epithelium under the anterior capsule is frequently seen in complicated cataracts, and Lamb [2] reported recently on the fibrous metaplasia of this proliferated epithelial tissue. This process may be so marked that the proliferated fibrous-like tissue protrudes quite far through the pupillary area. The tendency for such changes in complicated cataracts explains the frequent toughness of these capsules in doing intracapsular operation.

CONCLUSIONS

In cataracts caused by irradiation there is a tendency toward a proliferation of the epithelium under the anterior capsule into a metaplastic fibrous layer. This strengthens the anterior capsule and makes this type of cataract particularly suitable for intracapsular extraction. Extracapsular extraction in such cases is contraindicated because the lens epithelium remaining after the nucleus is extracted may continue to proliferate and form dense fibrous tissue, which tends to produce iridocyclitis and secondary glaucoma.

Some senile and some complicated cataracts also show a fibrous metaplasia of proliferated epithelium under the anterior capsule. When it is present it can be appreciated clinically even in the lesser degrees and is a factor in the successful removal of the lens in capsule.

The technical work was done by Miss Lily Kneiske, and the photomicrographs were made by Mr. N. E. Ross.

DISCUSSION

Dr. Arnold Knapp, New York: I want to congratulate Dr. Reese on the admirable results he has obtained in these difficult cases. I was particularly interested in noting the adequacy of extracapsular operation in the operative treatment of radiation cataract. Unfortunately, the intracapsular operation is not always successful. After 3 of the 6 extracapsular operations described by Dr. Reese, a complication consisting of a dense white pupillary membrane developed. This he regards as due to proliferation of the capsular epithelium, which in turn causes the

2. Lamb, H. D.: Anterior Capsular Cataract: An Example of True Metaplasia, Arch. Ophth. **17**:877 (May) 1937.

cyclitis and complications. My experience in these cases is limited, but I have been unfortunate enough to have a patient in whom this series of complications developed, and Dr. Reese was good enough to examine the patient with me.

In my case the white material developed in the pupil much too early for proliferation of the epithelium to have taken place. I therefore regard it as a retention of cortex, which perhaps in such cases is particularly toxic, and which I think leads to the cyclitis and to the dense pupillary membrane.

Dr. Grady Clay, Atlanta, Ga.: Results of animal experiments have varied so much with different animals and with different workers and there has been so little experimentation with controlled accurate dosage of radiation that such accumulated data are not altogether applicable to the human eye. However, in a general sense the lens of the infant seems to be susceptible to irradiation, showing an early disturbance of the epithelium, while the mature lens seems relatively radioresistant, the eyelids, conjunctivas and surrounding skin being much more sensitive.

During the past seven years I have used roentgen rays in the treatment of blepharitis and infected corneal ulcers, with most marvelous results. All the treatment was given in small suberythema doses (from 75 to 100 roentgens), the total not exceeding 1½ erythema doses. Corneas which appeared incurable have immediately begun to heal under such treatment. I therefore feel that roentgen irradiation is one of the most valuable forms of therapy. Recently I have examined many patients with these early disturbances with the slit lamp, with negative results.

FUSIONAL MOVEMENTS

ROLE OF PERIPHERAL RETINAL STIMULI

HERMANN M. BURIAN, M.D.
HANOVER, N. H.

Up to this time all investigations concerning the fusion of identical objects have been conducted with stimuli which had a simultaneous effect on the fovea of one eye and on certain surrounding areas located near the fovea of the other eye. Previously, no attempt had been made to learn how identical objects imaged on strictly peripheral disparate points of the retinas can affect the relative position of the two eyes.

For an investigation of this kind a haploscope, such as Bielschowsky and Hofmann used in their fundamental work on fusion, is inadequate. One needs an instrument which permits the stimulation of any desired areas of the two retinas with identical stimuli, the relative position, size, shape and brightness of which can be controlled.

An arrangement of several projection lanterns and polarizing material, which allows the dissociation of the two eyes, fulfils these conditions and has proved to be satisfactory.

Two projection lanterns are used to throw on an aluminized screen small test lines which can be arranged in a horizontal or vertical position (figs. 1 and 2). Polarizing material in front of the projection lanterns and also in front of each of the two apertures through which the observer looks (fig. 1) makes these test lines visible monocularly. The test lines can be presented horizontally or vertically and are so adjusted that they are on a level with the primary position of the observer's eyes. The measurement of the displacement of the test lines (i. e., of the relative position of the visual axes of the observer) produced by fusional stimuli is made by movements of one of the test lines with the aid of a vernier (not shown in the figure), which can be read directly to 0.01 of a degree and can be estimated to 0.003 of a degree. The settings can either be read directly from a scale or be marked with a punch on a strip of paper, so that a permanent record is obtained.

For the production of fusional innervations in small retinal areas, objects are used which consist of squares of varying size (fig. 2) which

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From the Dartmouth Eye Institute, Dartmouth Medical School.

Read before the Section on Ophthalmology at the Eighty-Ninth Annual Session of the American Medical Association, San Francisco, June 16, 1938.

Preliminary report of research done by members of the research group of the Dartmouth Eye Institute.

are projected on any desired place of the screen by a third projection lantern. The gap between the two test lines is fixated by the observer. These squares can also be presented either binocularly or in such a way that each square is seen only with one eye with the aid of polarizing material. With a double prism arrangement the images of the square can be separated up to any desired amount of disparity. By successive

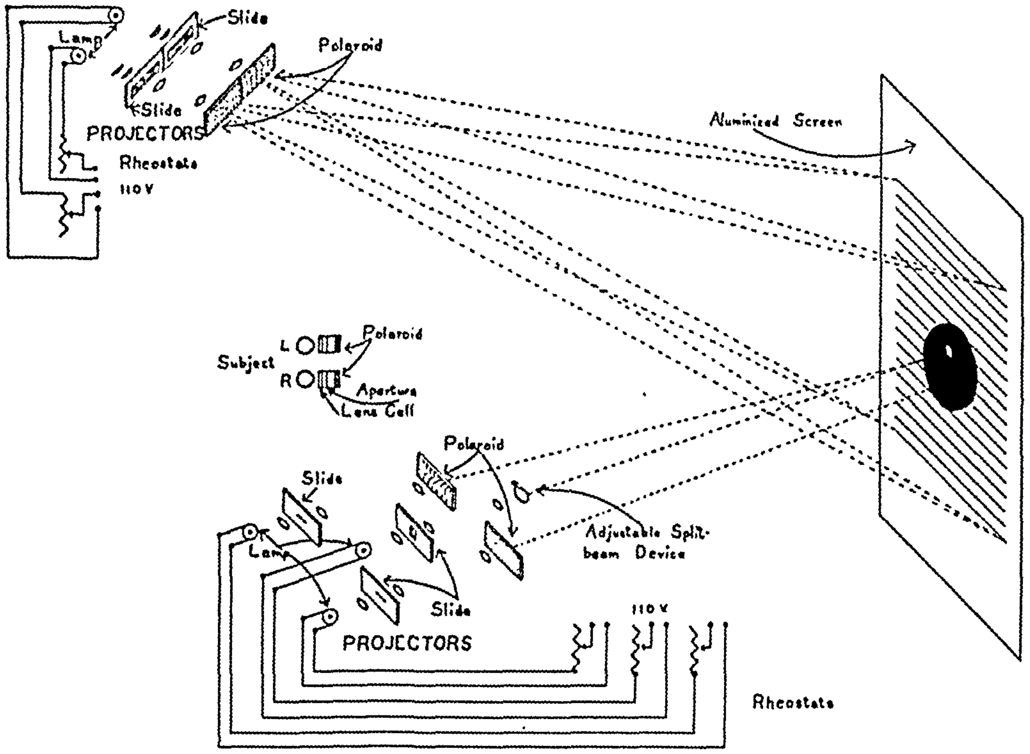

Fig. 1.—Diagram of the apparatus employed.

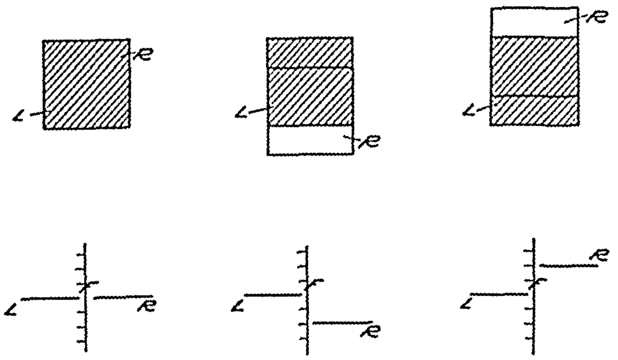

Fig. 2.—Diagram showing the influence of displaced peripheral stimuli (squares) on the position of the test lines.

stimulation of the various parts of the peripheral portion of the retina one can, so to speak, map out its fusional power.

If a greater area of the two retinas is to be stimulated, two further projection lanterns are used; these are also provided with polarizing material for the dissociation of the images of the two eyes. These

lanterns project on the screen identical images occupying a variable area of the visual field of the observer. Their use will be described later.

As was expected, with the arrangement in which the squares were used it was found that not only central but strictly peripheral stimuli are able to produce typical fusional movements. In other words, innervations to the extrinsic muscles arise whenever noncorresponding areas of the retinal periphery are stimulated within the limits of fusional amplitude. The type of innervation, i. e., whether a horizontal, vertical or cyclo movement of the eyes will result, is determined by the direction of the displacement of the stimulating images. The amount of stimulation exerted by the peripheral images depends on their intensity, in other words, the size and brightness, and on the distance from the fovea. In order to produce the maximum displacement of the visual lines or of the corresponding meridians allowed by the fusional amplitude, the stimuli must be larger the farther they are from the fovea. That the periphery plays an important role in producing fusional movements is demonstrated by the fact that stimulation of relatively small areas of the retinas, e. g., squares as small as 1 degree or 0.50 of a degree, at an angular distance of 12 degrees from the fovea, are able to produce noticeable fusional movements, although the peripheral images are necessarily so indistinct that the observer is unable to state whether or not they are fused.

Figure 2 represents a diagrammatic illustration of such an experiment. If the images of the two eyes are superimposed, the test lines will appear opposite each other to the observer; i. e., no vertical divergence has been induced. If, however, the square seen with the right eye is raised, the visual axis of the right eye will be raised accordingly and the right test line will therefore appear lower. The observer will have to raise the right test line in order to see again the two test lines opposite each other, as shown in figure 2. The inverse is true if the square seen by the right eye is lowered.

For the time being, only a displacement in the vertical direction has been used in the quantitative experiments, since it is well known that fusional experiments in this direction are much clearer and more precise and are less likely to be complicated by voluntary ocular movements than in the horizontal direction. The fusion movements due to peripheral stimuli, however, are also present in the horizontal direction and can be easily demonstrated.

The measurements obtained showed a high degree of accuracy. The mean deviations, as shown in the accompanying table, are within thousandths of a degree, while the settings of the test lines without any fusional object in the field are rather unsteady and at best within hundredths of an arc degree, owing to the continuous fluctuation of the innervations influencing the relative position of the two eyes.

It is well known that fusional innervations, produced by displacing one of the images from the macula to the periphery, persist for some time after the stimuli have been removed. Displaced peripheral images have an analogous effect, and in view of the great extent of the periphery it can be assumed that under normal conditions of seeing there is a definite preponderance of the peripheral fusional innervations on the position of the eyes relative to each other.

Figure 3 represents the results of an experiment in which the two peripheral images of a square with a side length of 1 degree, seen at 8 degrees above the fixation point, were displaced successively, the square belonging to the right eye first being raised, then lowered. The displacement of the squares is represented on the x axis and the correspond-

Average Scale Readings and Mean Deviations of Settings When Binocularly Seen Targets, 2 Degrees in Size and Separated Approximately 0.25 of a Degree, are Placed at Various Distances Above and Below the Fixation Point *

Location of Targets	Scale Reading†	Mean Deviation
12° above F. P.	— 0.332°	± 0.006°
8° above F. P.	— 0.334°	± 0.012°
4° above F. P.	— 0.372°	± 0.009°
2° above F. P.	— 0.362°	± 0.004°
2° below F. P.	— 0.293°	± 0.004°
4° below F. P.	— 0.311°	± 0.008°
8° below F. P.	— 0.326°	± 0.007°
12° below F. P.	— 0.348°	± 0.009°
Phoria (without fusion targets) beginning	— 0.215°	± 0.016°
Phoria (without fusion targets) ending	— 0.333°	± 0.044°

* Data for one subject (H. M. B.).
† Negative scale readings indicate positive vertical divergence.

ing vertical deviation of the eyes on the y axis. As can be seen, the amount of the vertical divergence is equal up to 0.75 of a degree to that of the displacement of the targets; from there on the ocular movements lag behind the displacement, until finally the stimuli no longer exert a fusional effect. The triangles indicate the phoria measurements, taken after the subject had regained central fusion.

One of the most important results shown by these experiments is that peripheral retinal stimuli are strong enough to break the fusion of images situated on corresponding areas of the macular region, provided they cover a sufficient area; the images will appear double and cannot be fused as long as the displaced peripheral retinal stimuli are present, no matter how hard the observer may try to maintain central fusion. This can be shown by the following arrangement: Identical print is projected from two lanterns, and the images are superimposed (fig. 4 *A*); on a central black disk, the size of which can be varied, the two

test lines mentioned before appear opposite each other, and a central fusional object seen binocularly (square) will appear single. As soon as the image of the print corresponding to one eye is raised, the fixation object immediately appears double, and the test lines are displaced, as shown in figure 4 *B*. This breaking up of the central fusion of squares as large as 1 degree occurs even if the central black disk has a diameter

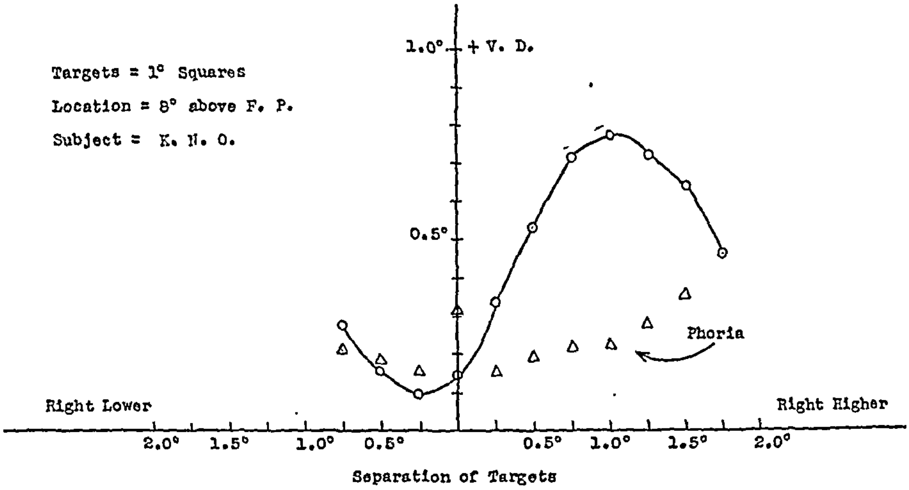

Fig. 3.—Data plotted from one experiment, showing the influence of a displaced peripheral stimulus on the position of the eyes.

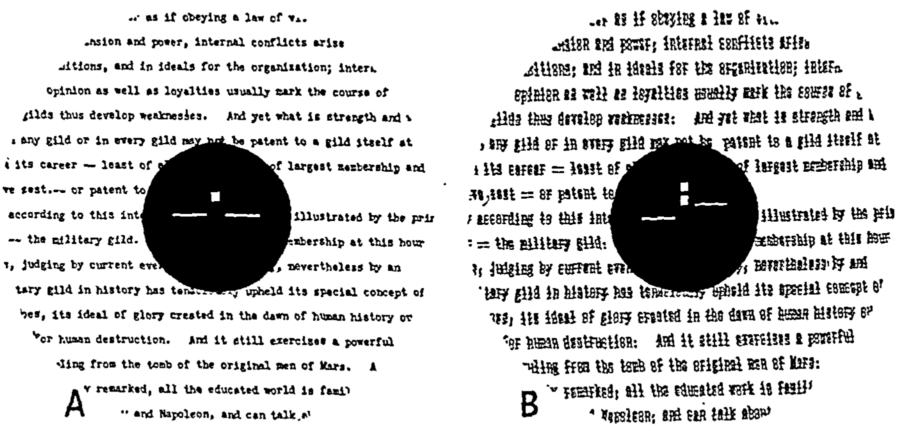

Fig. 4.—Diagram showing the doubling of a centrally fixated object (square) by displacement of peripheral images (print). *A*, print superimposed for both eyes. *B*, print displaced (higher in the right eye).

of 5 arc degrees, leaving an annular pattern of 7 arc degrees in the periphery; this is true even if only a half, or merely a quarter, of the peripheral field is exhibited.

If, as appears from the foregoing presentation, powerful fusional stimuli are exerted by peripheral retinal areas, and these stimuli affect the relative position of the eyes and, under certain conditions, may even

cause a loss of central fusion, one is faced with a problem which not only is interesting theoretically but is of great significance from a practical point of view. Theoretically, it shows that the functions of the peripheral portion of the retina, to which only twilight vision and the perception of motion have been ascribed heretofore, play an important part in the process of fusion. It is, however, the practical aspect of the problem which should be emphasized.

Every ophthalmologist knows patients who are not relieved by a most painstaking correction of refractive errors and muscle imbalances or of possible symmetric differences in the size and/or shape of ocular images. On the basis of experimental as well as clinical experience, one is warranted in assuming that there are persons with a relative asymmetry of the images of the two eyes. It is evident that such persons will never be able to fuse simultaneously central and peripheral images and that therefore conflicting innervations and considerable discomfort must arise, unless the patient is able to suppress either the peripheral or the central part of the images.

PRECOCIOUS CATARACTS AND SCLERODERMA (ROTHMUND'S SYNDROME; WERNER'S SYNDROME)

REPORT OF A CASE

SIGMUND A. AGATSTON, M.D.
AND
SAMUEL GARTNER, M.D.
NEW YORK

Thirty-two cases of this syndrome have been reported to date. In 1868 Rothmund first described the combination of a cutaneous condition which appears to have been scleroderma with precocious development of bilateral cataract. He reported 5 cases of this syndrome in the same family; 3 other members of the family presented only cutaneous changes. The patients were all young children. Since this report the disease has been identified as Rothmund's syndrome. Werner in 1904 described 4 such cases in 1 family, definitely designating the cutaneous condition as scleroderma. In 1934 Oppenheimer and Kugel reported 2 cases of this condition, which they called Werner's syndrome.

In 1931 H. Mamou reviewed the subject carefully and collected reports of 30 cases from the literature. He suggested parathyroid dysfunction as the basic cause.

This disease is a hereditary and familial disorder characterized by precocious development of bilateral cataract, early graying of the hair, premature senility and a variety of endocrine disturbances. Many of the cases showed marked similarity. The history of consanguinity is common. The syndrome generally occurs in family groups, though some isolated cases have been reported. Rothmund's original cases occurred in 3 families. In 2 of these families 2 brothers married 2 sisters. Two of the children from each of these marriages were affected. In the third family cousins married, and 1 child was affected. Guillain in 1923 described the cases of 2 sisters. Von Arady in 1927 described a case in which the parents of the patient were first cousins. Four cases were reported by Krebs, Hartmann and Thiébaut; 3 of the patients were siblings, and 1 was an aunt. Nine other members of the family had minor developments of the same syndrome. The grandparents were first cousins. The cases reported by Oppenheimer and Kugel were

From Dr. S. A. Agatston's Ophthalmic Service at the Montefiore Hospital.

those of 2 brothers. Their parents were first cousins and so were their grandparents. In our case the parents were first cousins.

The defect appears to be a recessive characteristic that is particularly influenced by consanguinity.

The cataracts are bilateral, though one may be somewhat more advanced than the other. Opacities of the lens usually appear after the age of 30, though they have been reported as early as the age of 4 years by Rothmund. The cataracts are cortical and mature within a few months. There is no pathologic involvement of the retina or the optic nerve. The association of endocrine disorders with cataracts is not unknown. The cataracts reported in these cases with scleroderma are similar to those reported in association with endocrine disorders. The fetal nucleus is usually clear; the opacities develop in the cortex. None of the patients had any intraocular disease which could account for secondary cataracts. Mamou in 1931 made careful slit lamp examinations and supplied interesting drawings of eyes with cortical and subcapsular opacities.

The graying of the hair appears early in life and has been described at as early an age as 8 years. Alopecia develops early. The patients look much older than their age.

The skin shows the changes of scleroderma, particularly the face and the extremities. Chronic ulcers of the feet and toes are common. Poikilodermatous changes and telangiectasis have also been noted. Laryngeal involvement often occurs with change of voice, which is usually high pitched.

The build is characterized by a normal trunk and small extremities, while the face and head look senile.

The endocrine disturbances are varied. The gonads are usually affected, and deficiency is the rule. In the female delayed and irregular menstruation occur. In 1 case menstruation ceased at the age of 16. In the male the genitals are usually underdeveloped, and there are feminine characteristics. Potency is usual, but fertility is fortunately low. Thyroid disturbance has occurred in a number of cases, with the development of exophthalmic goiter. Alteration of the calcium metabolism has been noted. One of Oppenheimer and Kugel's patients had a negative calcium balance. They suggested the possibility of parathyroid disturbance to account for this. Von Arady associated the disease with a pituitary disorder. Diabetes, which may be a pituitary or pancreatic disorder, has been encountered.

Early arteriosclerosis and hypertension have been noted in several cases. Roentgenograms have shown calcification of many arteries. Our patient had hypertension, and roentgen examination showed calcified blood vessels in the lower extremities at the age of 26.

Our patient also had a malignant tumor on his forearm, a fibroliposarcoma. This is the only case in the literature in which a malignant growth is added to the rest of the syndrome.

The number of reported cases in which the condition is fully developed is relatively small. However, in many of the case reports mention is made of other members of the family with minor or incomplete development of this syndrome. Some have only premature graying of the hair; others, precocious cataracts or only scleroderma. In such cases the condition would ordinarily escape diagnosis. The association of premature graying of the hair and cataracts in relatively young persons has been known to ophthalmologists.

REPORT OF CASE

History.—H. T., a Jewish bookkeeper aged 38, born in Hungary, was seen at the Montefiore Hospital. Until the age of 23 he was well and active. At the age of 23 his hair began to fall out. When he reached the age of 30 his hair was gray.

At the age of 31 he noticed hazy vision, and early bilateral cataract was diagnosed. It matured rapidly. When he was 31 iridectomy was done on both eyes at the New York Eye and Ear Infirmary by Dr. M. L. Berliner. When he was 37 the cataract of the left eye was removed at the New York Eye and Ear Infirmary by Dr. M. L. Berliner, and when he was 38 that in the right eye was removed at the Montefiore Hospital by one of us (S. A. A.).

An ulcer on the patient's foot developed when he was 26. This was attended at the Hospital for Joint Diseases. Roentgen examination at that time showed calcification of the posterior tibial artery and a severe circulatory disturbance. At another institution a diagnosis of thromboangiitis obliterans was made.

When the patient was 37 a lump developed on the left forearm and grew rapidly. Roentgen examination showed that the neoplasm had destroyed the radius. The growth was removed at the Lincoln Hospital. A pathologic diagnosis of fibroliposarcoma was made. The wound healed well. When the patient was last seen no recurrence of metastases had been noted.

At the age of 36 the patient's left ring finger was injured and gangrene developed at the tip. Amputation of two distal phalanges was done at the Bellevue Hospital. The stump later became gangrenous and necessitated another operation at the Morrisania Hospital.

Starting at the age of 33, the patient's teeth broke readily, and several broke while he was chewing. Some teeth loosened and fell out spontaneously.

The patient's habits were always temperate. He occasionally smoked, never indulged in alcohol and drank about one cup of coffee a day. He had tonsillitis in 1932, following which tonsillectomy was performed.

Family History.—The patient was married and had a son of 9 years, who appeared to be normal. His mother and father were first cousins. His mother had hay fever. His father died at the age of 35 of a tumor of the neck. His mother was alive and well at the age of 73. Two brothers died in infancy, the cause of death being unknown. He had no sisters. His father's sister married her first cousin. Their daughter, aged 33, has very small extremities and another daughter is normal.

Examination.—When the patient was examined at the age of 38 he had the appearance of a man between 55 and 60. Most of his head was bald, and

the remaining hairs were gray. His mentality was normal and his memory good. He was 5 feet and 2 inches tall (157 cm.) and weighed 115 pounds (52 Kg.). His limbs were small, and he wore size 4½ shoe and size 6¼ glove. His measurements were as follows: from the anterior superior iliac spine to the external malleolus, 31½ inches (80 cm.) on each side; from the acromium to the tip of the middle finger, 24½ inches (62 cm.) on the right and 25½ (65 cm.) on the left; circumference of the chest, 35 inches (89 cm.); circumference of the right ankle, 6¼ inches (15.9 cm.); circumference of the waist, 35 inches (89 cm.) and circumference of the wrist, 5½ inches (14 cm.). He had a delicate build. The skin over his face and extremities was taut and brownish.

Both eyes had postoperative colobomas of the iris above. The right eye had cataract, which matured in a few months. When the patient was first seen he had postcortical opacities, some subcapsular vacuoles and anterior peripheral cortical opacities. The cataract was removed on Oct. 10, 1938. The postoperative result was good. The fundus was normal with the exception of senile arteriolar sclerosis, with slight hypertensive changes. The left lens had been removed previously. There was a delicate secondary membrane. The fundus was visible but hazy. With a + 10.00 D. sph. $\supset$ + 4.50 cyl., axis 120° vision in the left eye was 15/50.

Examination of the teeth showed many to be broken off at the edge of the gum. The roots were carious. Several teeth were missing, and a few were in good condition.

The thyroid was not palpable.

The trunk had a normal build. The configuration of the pubis and the arrangement of the pubic hairs were feminine. The penis and scrotum were small. The breasts were normal. The heart and lungs were normal. The blood pressure was 182 systolic and 102 diastolic.

The extremities were small, and the hands and feet were extremely small. The left ring finger had been amputated at the first phalanx. The stump was well healed. Over the left forearm there was a recent postoperative scar. The skin over the forehead, feet and hands was tight, thickened and dry. Over both legs and feet there were light brown spots. There was an ulcer over the left achilles tendon. Neurologic examination gave negative results. Normal reflexes were present; no abnormal ones were found.

Laboratory Report.—Urinalysis gave negative results. The Wassermann reaction was negative. The blood count showed 5,950,000 red cells and 5,750 white cells, with 65 per cent polymorphonuclears and 35 per cent lymphocytes. Chemical analysis of the blood revealed 87 mg. of sugar per hundred cubic centimeters, 154 mg. of cholesterol, 33 mg. of nonprotein nitrogen and 12 mg. of urea nitrogen. The sugar tolerance test gave normal results. A roentgenogram showed calcification of the posterior tibial arteries.

SUMMARY

A syndrome is presented characterized by the appearance in relatively young persons of bilateral cataract, early graying of the hair, scleroderma and various endocrine disorders. The literature is reviewed and a case reported.

Underdevelopment and hypofunction of the gonads are usual. Parathyroid disturbance has been suspected to be a factor. Some patients have had hyperthyroidism; others diabetes.

The early development of arteriosclerosis has been noted, with hypertension in some cases.

The build of patients with this syndrome is characterized by a normal size trunk and small extremities. The general appearance is that of premature senility. The patients look from 20 to 30 years older than their actual age.

The disorder is inherited, and the cases have occurred mainly in family groups. A history of consanguinity has been found in most cases. It is apparently a recessive characteristic.

In addition to the other findings, our patient had fibroliposarcoma.

56 East Eighty-Ninth Street.

1749 Grand Concourse.

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(*a*) LEIOMYOMA AND (*b*) HEMATOMA OF THE IRIS

REPORT OF CASES

E. C. ELLETT, M.D.
MEMPHIS, TENN.

(*a*) CASE OF LEIOMYOMA OF THE IRIS

R. T., a white man aged 47, was referred to me by Dr. W. N. Reed, of Amory, Miss., on March 16, 1937. He said that for twenty years he had noticed that the pupil of his right eye was elongated instead of round and that about fifteen years ago something seemed to be growing into the anterior chamber. This was of a light color at first; later it grew darker, and about three weeks before he was seen by me a hemorrhage had occurred into the front of the eye. The blood absorbed, leaving the appearance presented by the patient on March 16.

The left eye was normal in every respect. Tension equaled 15. Vision in the right eye was 6/18, and the patient was able to read Jaeger's test type 16: with a + 2.00 cylinder, axis 165, vision was 6/5, and with the addition of a + 2.00 sphere he could read Jaeger's test type 1. The tension was 15. The pupil was free and active except where the growth involved the iris. The lesion, apparently a new growth, involved the whole thickness of the iris from the pupil to the base and lay in the lower nasal quadrant. It was gelatinous and semitransparent, with two hemorrhagic spots at the apex of the triangular growth, and vessels could be seen in its substance. There was a slight opacity of the lens behind the growth. The media were clear and the fundus normal. The appearance is well shown in figure 1 *A*.

As the growth extended to the root of the iris, and possibly further, and as its appearance was exactly that of a sarcoma of the iris, its removal was advised. Its probable nature and characteristics were explained, and the opinion was expressed that complete removal of the tumor would not be possible and that unless it was completely removed it would recur and extend. The patient decided to have the eye removed, which was done on March 20. A glass ball was implanted, and healing was prompt. On Jan. 11, 1938, the patient reported himself in good health. The eye was sent to the Army Medical Museum, which in turn submitted the section to Dr. Verhoeff. His report follows:

"*Microscopic.*—I believe the tumor to be an epiblastic leiomyoma of the iris. Beneath the pigment epithelium the cells often closely resemble those of the normal dilator and sphincter muscles, and I feel that if any special staining could be used, myoglia fibrils could be demonstrated here. Elsewhere the tumor cells become so atypical that their nature cannot be determined. In places the tissue closely resembles neuroglia, but this is consistent with my assumption that the tumor arises from the neural epithelium, just as does the dilator muscle. The large nuclei with

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Read at the Seventy-Fourth Annual Meeting of the American Ophthalmological Society, San Francisco, June 10, 1938.

vacuoles are interesting but of no diagnostic significance. I have sections of a similar but much smaller iris tumor of the same nature, which I believe to be an epiblastic myoma. I have not reported this because I have no conclusive proof as to its origin."

Two years ago Frost [1] reported a case of leiomyoma of the iris to this society. Verhoeff [2] has reported 1 case, and the literature on leiomyoma, both of the iris and the ciliary body, is covered in these two papers. I have not been able to find an additional case reported in the literature; so I shall refer to those two papers for references to the subject.

Collins and Mayou [3] called attention to the facts that the muscle fibers of the iris are derived from the neural epiblast, while those of the ciliary body are mesoblastic, and that tumors composed of what have been considered to be unstriped muscle fibers are described as arising from each of these divisions of the uveal tract. The points relied on for diagnosing a tumor as a myoma rather than a spindle cell sarcoma are: the point of origin, the slow growth, the similarity of the cells to those of unstriped muscle and the rod-shaped nuclei. The special stain used by Verhoeff, Mallory's phosphotungstic acid-hematoxylin, will show the myoglia fibrils, which are more convincing and characteristic than the other features.

The case reported by Van Duyse, which according to Verhoeff was probably one of myoma, resembled my case in that the growth was of long duration, twenty-one years, and repeated hemorrhages occurred in the anterior chamber. Glaucoma was not present, though it was in Helleberg's case.

Better laboratory technic, especially better staining methods, might show more cases of leiomyoma among the rather numerous cases of nonpigmented sarcoma of the iris that have been reported.

It is hard to be sure in regard to the malignancy of these tumors. Clinically, they resemble sarcoma so closely that one could hardly be sure, and even if they were benign, their growth would eventually destroy the eye if they were left alone.

(b) CASE OF HEMATOMA OF THE IRIS

T. B. A., a white man aged 53, was seen on Dec. 21, 1937, through the courtesy of Dr. H. J. Kellum, of Tupelo, Miss., with a history that in the previous September he had noticed a dark spot on the right iris and that the eye had been inflamed most of the time since the spot appeared. Dr. Kellum found a small tumor on the iris, down and out, and shortly after his first examination there was a hemorrhage in

1. Frost, A. D.: Am. J. Ophth. **20**:347, 1937.
2. Verhoeff, F. H.: Arch. Ophth. **52**:132, 1923.
3. Collins, E. T., and Mayou, M. S.: Pathology and Bacteriology of the Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1925.

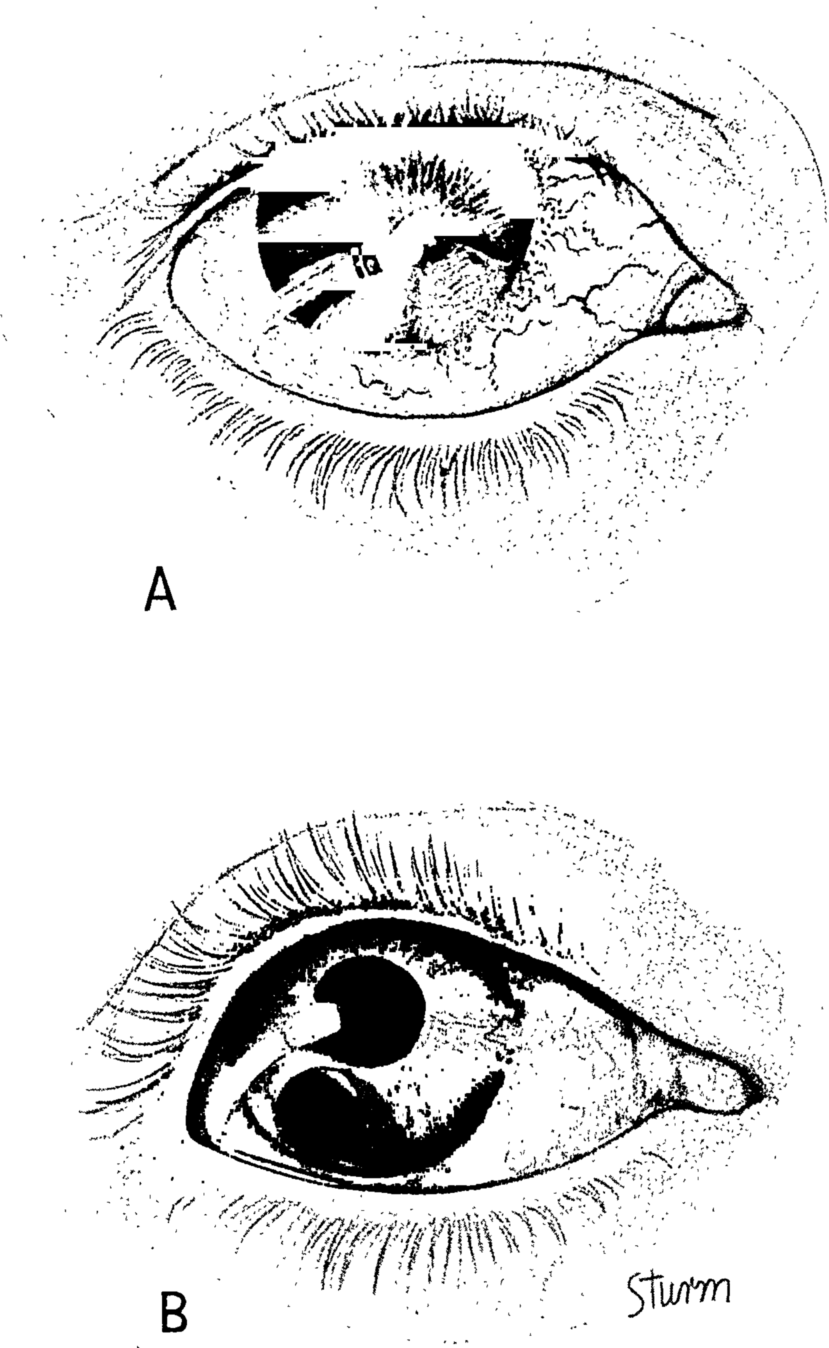


Fig. 1.—*A*, leiomyoma of the iris. *B*, hematoma of the iris.

the anterior chamber, obscuring the original lesion and spreading in the lower part of the chamber. The left eye was normal except for a small pterygium, and the vision was normal with a +0.75 D.

The right eye also showed a small pterygium. On the iris, down and out, was a small rounded tumor about 5 mm. in diameter, projecting from the anterior surface of the iris and touching the cornea. The tumor was dark red and smooth and had the appearance of a cyst filled with blood. At the upper border the wall of a cyst could be made out, not in contact with the blood, as if the cyst was not quite full of blood. Down and in, the wall of the cyst seemed to have ruptured, and the blood had passed into the anterior chamber. The vision was 6/10 (partly) and was not improved by glasses. The fundus was well seen and normal, and tension was 18. The growth did not reach either the pupillary margin or the root of the iris, and its appearance is well shown in figure 1 *B*.

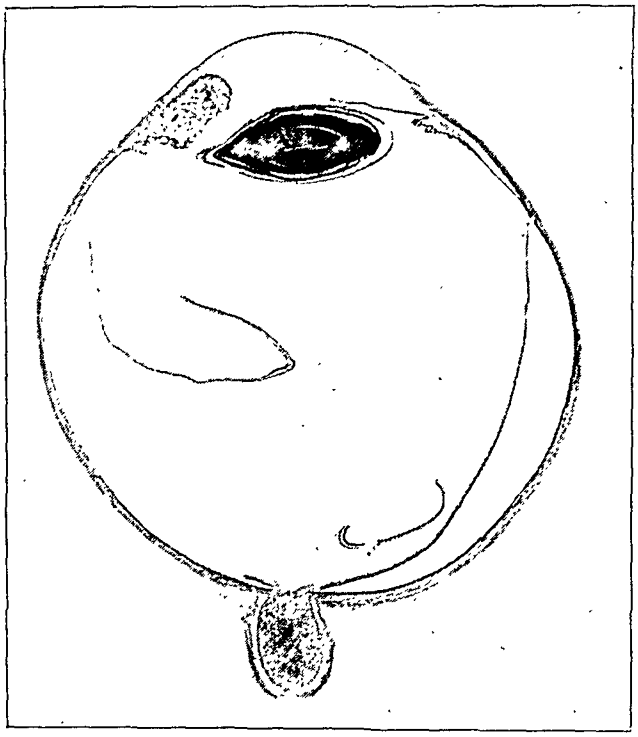


Fig. 2.—Leiomyoma of the iris.

On December 28 a keratome incision was made just above the tumor, at the limbus, and the incision was enlarged downward past the base of the tumor with Stevens' scissors. The growth was grasped with iris forceps, drawn out and removed, together with iris tissue on each side and beyond the base of the growth. The eye was dressed in the usual manner. The chamber promptly reformed, and the patient was dismissed from the hospital on the third day. Reports from him are to the effect that he has had no further trouble.

The specimen was sent to the Army Medical Museum, where the following report was made:

"*Gross.*—A small dark nodule attached to a thin dark membrane.

"*Microscopic.*—The section shows iris tissue, in one portion of which there is a large circumscribed hemorrhage forming a nodule on the anterior surface. Anteriorly, the hemorrhage had almost broken through into the anterior chamber. The red blood cells in the hemorrhage show various stages of degeneration, but nowhere does the hemorrhage look more than several days old. Around the edge there is a slight leukocytic and round cell infiltration, with a predominance of

eosinophils. The vessels of the iris are not particularly sclerotic. There is a scattering of lymphocytes through the iris stroma. There is no evidence of a neoplastic growth.

"Diagnosis.—Hematoma, recent, of iris."

In reply to a question as to the possibility of a small growth at the base of the tumor, a supplementary report was later received as follows:

"All of the block that we received from your case T. B. A. was embedded in paraffin. Following the receipt of your letter, microscopic sections were taken from the deeper portion of the block; these show a similar picture.

"No evidence of neoplastic tissue is found."

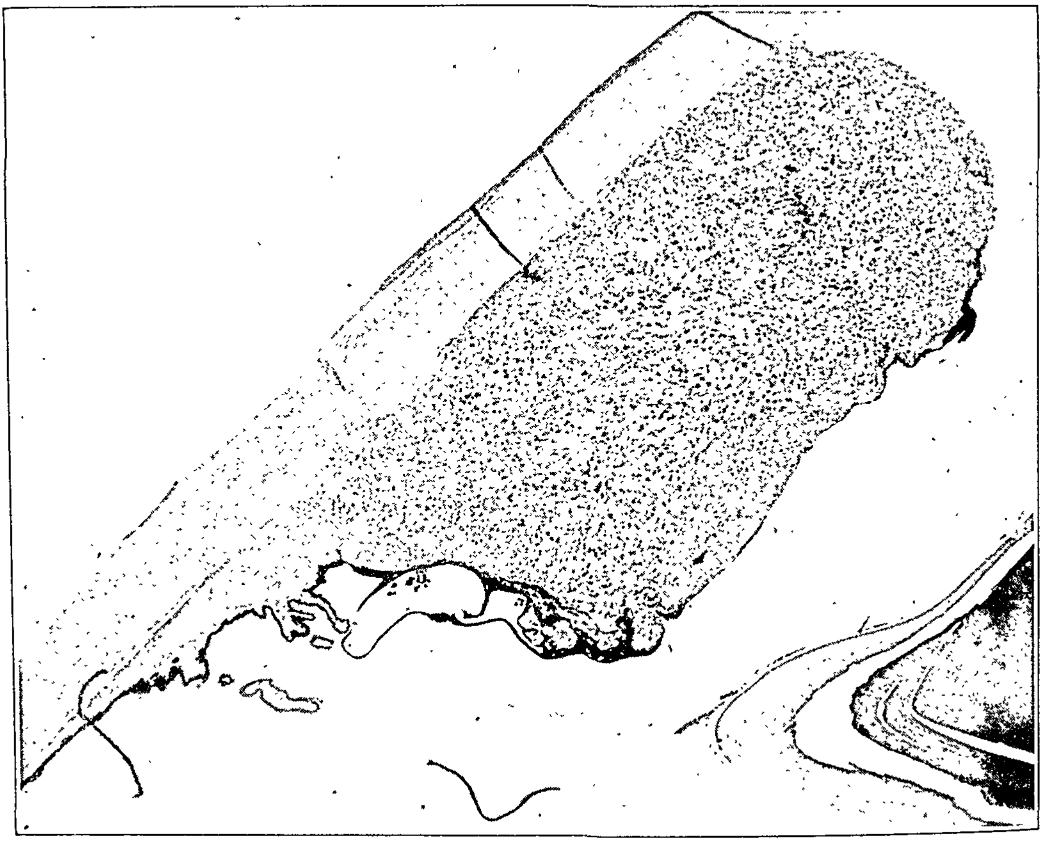


Fig. 3.—Leiomyoma of the iris.

Cysts of the iris are classified by Parsons [4] into the following groups: (1) implantation cysts, including pearl cysts and atheromatous cysts; (2) retention cysts; (3) congenital cysts; (4) cysts of the retinal epithelium, and (5) parasitic cysts.

According to this classification, the present case is one of retention cyst, into which a hemorrhage had occurred. The absence of a definite cyst wall would suggest the formation of the cyst in accordance with

4. Parsons, J. H.: The Pathology of the Eye, New York, G. P. Putnam's Sons, 1904, vol. 1, pt. 1, p. 311.

the idea of Schmidt-Rimpler, that is, that they are due to closure of one or more of the crypts of the iris.

Collins and Mayou [5] described cysts with an endothelial lining as being found in the iris. In this case there is no endothelial lining. They agreed with Schmidt-Rimpler that these cysts are due to blocking of the mouths of the crypts of the iris from fusion of the thickened strands which are often seen crossing them. They may form after an injury.

Fig. 4.—Hematoma of the iris.

Nadal [6] removed a congenital cyst, measuring 6 by 8 mm., from the iris of a boy of 11 years of age. The cyst was lined with epithelial cells, which would appear to be in conflict with the idea of Schmidt-Rimpler as to their origin.

There seems to be no reported case exactly like this one, if I have correctly interpreted it, at least in the literature to which I have access.

5. Collins and Mayou,[3] p. 211.

6. Nadal, R.: Arch. d'opht. **31**:363, 1911.

The view was taken that there was a small tumor from which a hemorrhage had occurred, and, like most tumors of the uveal tract, it was thought to be probably sarcomatous. As it appeared possible to remove it completely by iridectomy, it was thought best to attempt this without delay. Though no tumor was found to be present, the removal of the cyst was justifiable.

Fig. 5.—Hematoma partly encircled (on the side nearest the pigment epithelium) by a large bifurcating vessel of varying diameter.

DISCUSSION

Dr. Georgiana Dvorak-Theobald: I have had the opportunity of examining sections from each growth. I agree with Dr. Verhoeff that the growth in the first case is a leiomyoma.

Authors generally agree that it is at times difficult to distinguish a leiomyoma from a spindle cell sarcoma or a cellular fibroma. Differential staining should always be done. Vacuolated cells and giant cells are sometimes found. In describing leiomyoma of the urinary bladder,

Caylor and Walters described the vacuolated cells as "tadpole-shaped cells" and stated that the nuclei of the giant cells are clearly myoblasts.

In speaking of leiomyoma, Ewing stated that in actively growing tumors there is an increase in the neoplastic character of the cells, especially in their size, and in malignant leiomyoma large spindle cells appear and giant cells of enormous dimensions form. In the specimen under discussion, the more mature cells continue forward from the ciliary muscle, and the malignant cells appear toward the pupillary margin (fig. 3).

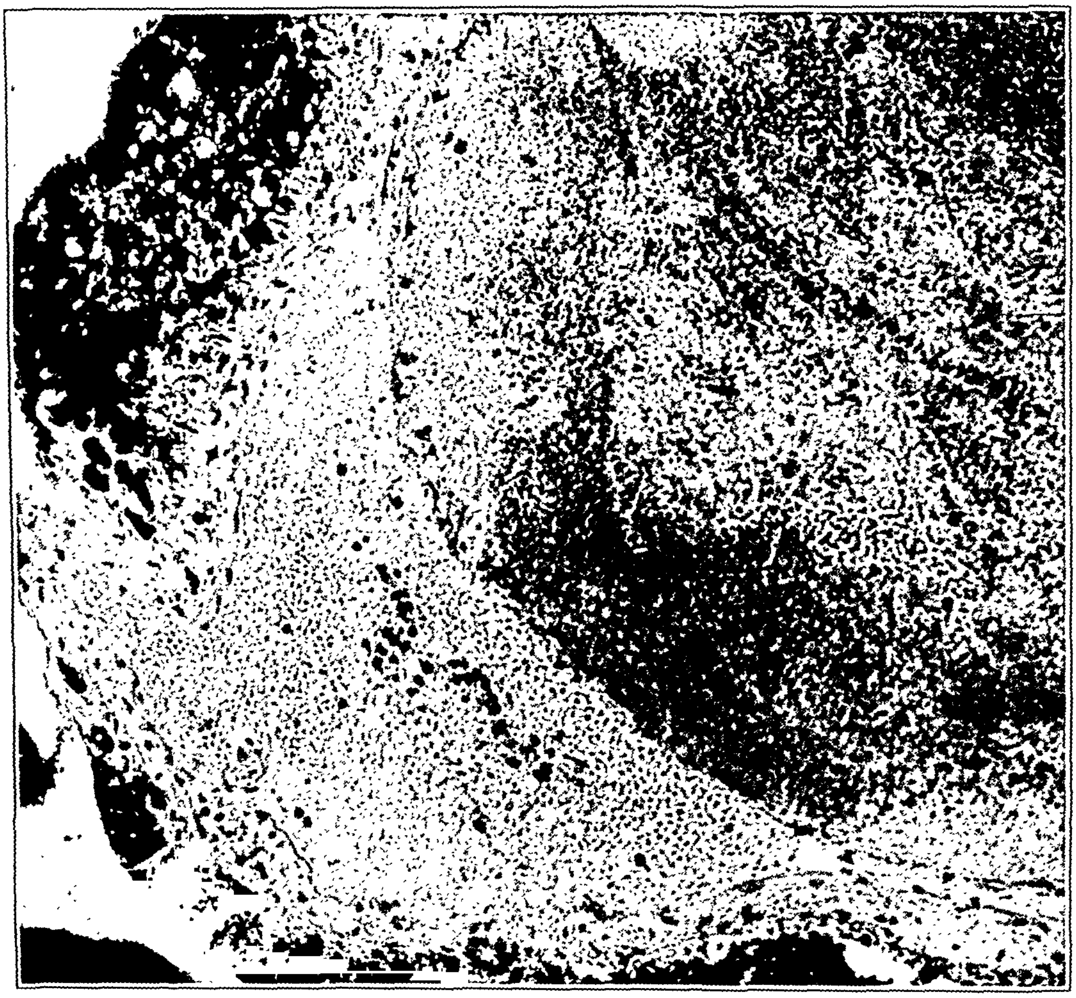


Fig. 6.—Marked dilatation of the iris vessel which bounds the hematoma on one side.

The growth in the second case, which had the appearance of a blood cyst, should be classed as a hematoma. The walls of a cyst are lined with epithelium, but the walls of this tumefaction consisted of iris tissue. The literature on cysts of the iris is loose and confusing and should not be further complicated. Dr. Ellett's case is certainly unusual and well brought to the notice of the members of the society. Hematoma does not occur without cause; in this case there must have been some condition that led to it. The patient noticed a dark spot on the iris three months before operation; the pathologic report states that "nowhere does the hemorrhage look more than several days old."

This would lead one to believe that some other condition preceded the hemorrhage. Figure 5 clearly shows a large bifurcating vessel of varying diameters partly encircling the hematoma, on the side nearest the pigment epithelium. This may have been an aneurysm, angiectasis or an angioma. Figures 6 and 7 illustrate the features to which attention is called.

Dr. Albert D. Frost, Columbus, Ohio: In such cases it is impossible to make a clinical differentiation of leiomyoma from sarcoma. The diagnosis can be made as the result of histologic study, and even then it may be difficult to determine definitely whether the tumor originates in smooth muscle fibers.

I had a patient, a doctor's wife, who had a tumor of the iris. I had done refraction for her within a year previous to the time she consulted me for the condition in the iris, and there had been no evidence of a tumor. She was observed from August 1934, when the growth was first noted, until March 1935, at which time enucleation was done. The tumor was evidently of more rapid growth than that which Dr. Ellett described, and it was more diffusely scattered through the tissue of the iris.

Fig. 7.—Hematoma of the iris.

One thing of particular interest was the ectropion of the uveal pigment at the pupillary margin. One wonders whether the smooth muscle cells still retain their contractility in order to produce this ectropion.

The patient had vision of 20/20 in the involved eye, and when the question of a tumor arose she became anxious and apprehensive. Although she has lost a seeing eye and knows that the tumor was not malignant in that it has not produced metastases, she is happy to have had the eye removed and has been well ever since.

THE FUNDUS OCULI IN GENERALIZED HYPERTENSION AND ARTERIOSCLEROSIS

ROBERT SALUS, M.D.
Professor of Ophthalmology, German University of Prague
PRAGUE, CZECHOSLOVAKIA

TRANSLATED BY ERNST WALDSTEIN, M.D., NEW YORK

The changes in the retinal vessels have been studied with peculiar attention since the beginning of clinical ophthalmology, as the background of the eye is the only place in the body where small arteries and veins, precapillaries and even capillaries can be observed covered only by a crystal clear tissue, in their natural position and color and magnified. Because of these favorable circumstances, important conclusions as to the pathology of the circulatory apparatus were naturally expected from such examinations. The importance of such conclusions, however, has increased considerably, owing to the knowledge that an almost complete parallelism exists between the retinal vessels and the cerebral vessels and that therefore one has an undisputed right to draw conclusions as to the condition of the cerebral vessels on the basis of changes in the vessels of the fundus.

I shall discuss briefly these changes.

The alterations of pure arteriosclerosis, not combined with hypertension, are, in accordance with the underlying pathologic process, not frequent. In this category belong fatty or chalklike glistening circumscribed alterations of the arterial walls, irregularities of the caliber because of aneurysmatic or rosary-like formations, opaqueness, thickening of the arterial walls, increased brilliancy of the vascular reflexes and tortuosity of the arteries.

Diagnosis is easy only in the advanced stages; in the beginning stages the diagnostic value of the changes just mentioned is not uniform and not unconditional. It is therefore not advisable to commit oneself as to the diagnosis before several, and especially the first enumerated, variations have made their appearance. The diagnosis of arteriosclerosis is by no means justified by the existence of tortuous vessels or somewhat more accentuated reflexes; such alterations may be due to other causes and are frequently congenital.

One is able to make a diagnosis of general hypertension and the diseases connected with it more definitely from an examination of the fundus.

The first symptom which proves with absolute certainty the diagnosis of generalized hypertension is one which I have called crossing arch (dubbed frequently "Salus' symptom"). To describe it briefly: where an artery crosses a vein, the latter forms before and behind the crossing an arch, more or less large, and within the limits of this arch appears somewhat attenuated or even totally or partially invisible.

The cognition of this phenomenon is credited to Marcus Gunn, who was the first to describe it in 1892. His explanation of the phenomenon was generally rejected; thus his communication was quickly forgotten, since hardly anything was known about the essence and the importance of hypertension at that time. More than thirty years later I directed attention to this almost forgotten and yet important symptom and presented an explanation for its genesis; this explanation was later confirmed by H. Friedenwald and Guist by anatomic studies. Since then, along with a number of ophthalmologists, I have found that the symptom has a certain importance in its clinical, diagnostic, pathologic and prognostic aspect which exceeds its purely diagnostic value.

In regard to this phenomenon, two points are chiefly of interest here: The crossing arch is absolute proof of hypertension. It is missing only in a small number of cases of permanent hypertension, chiefly for anatomic reasons (either the larger vessels do not happen to cross or the vein lies above the artery at the crossing); it is much rarer in intermittent hypertension and appears chiefly in the cases in which the condition has a tendency to become permanent.

Some English and American authors go too far in asserting that one can gage the height of the blood pressure directly by the evolvement of the crossing variations and that differences of 10 or 20 mm. of mercury can be estimated in that way. Just the same, and I previously emphasized this point in my publication of 1928, one can safely assume that, taken all in all, the changes are more marked the more evident the hypertension and the longer it exists. This fact can, of course, be utilized prognostically. In 1934 Brana and Radnai with a series of 100 hypertonic persons studied the question of whether, and on the basis of what changes, an individual prognosis quoad vitam could be made. They studied practically everything relevant to the question, i. e., the height of the systolic and the diastolic blood pressure, the electrocardiographic picture and the eyeground, and found that the most definite conclusions could be drawn from the crossing phenomenon. The mortality from hypertension when this phenomenon was absent or just noticeable was 2 per cent within one year; in cases in which marked variations in the phenomenon occurred, 60 per cent, and in cases of the most severe involvement, 100 per cent.

Besides the crossing phenomenon, there is in the fundus still another group of changes from which one is able to infer the existence of general hypertension. To this group belongs the attenuation of the peripheral arteries, vessels that correspond to arterioles. This change (Salus, 1931) is diagnostically less important; theoretically, however, it is interesting, since it demonstrates how the pathologic process (permanent spasm or thickening of the vascular wall, a question that cannot as a rule be decided by ophthalmoscopic examination) starts in the periphery or in the capillaries or precapillaries and pushes centralward while the disease progresses. Thus one sees occasionally the aforementioned attenuation in a marked degree in the very early stage while the main branches of the central artery are still wide, perhaps even somewhat dilated; in the far advanced stage the latter, too, are more or less thinner and, finally, markedly thinner.

Of other changes that belong in this class, the most important are the dazzling reflexes of the arteries (Gunn, 1892), the so-called silver wire arteries, an infrequent symptom but characteristic and present only in the advanced stages. Another change is the tortuosity of the veins, especially of the finest visible venules (Guist).

So-called albuminuric retinitis is also a symptom of hypertension to a certain extent. I shall therefore discuss it because of a peculiar importance which has been ascribed to it for the prognosis quoad vitam for some time.

As a matter of fact, it can still be said that the diagnosis of retinitis albuminurica means a death sentence, to be fulfilled within two years. The advances in the treatment of hypertension in recent years, connected chiefly with the name of Volhard, have so far hardly brought about any change in this respect. One should, however, note that this fatal prognosis for albuminuric retinitis holds good only in cases associated with malignant nephrosclerosis; that it is not valid in cases of retinitis associated with toxic glomerulonephritis (e. g., during pregnancy or after scarlet fever) which, along with the underlying disease, may be cured, and that it is not applicable, at least not in the way mentioned previously, which circumscribes the time limit, to cases of typical retinitis associated with hypertension which are clinically identical except for an intact renal function. If I have stated previously that the changes of the retinal vessels correspond to the degree and the duration of hypertension, I now have to modify this statement to a certain degree.

There are cases in which the general examination reveals hardly anything of importance except a moderate degree of hypertension but the fundi show crossing alterations of high degree and occasionally beginning retinitis and, strange to say, even marked retinitis. Some

of the patients who could be followed died within quite a few years of cerebral apoplexy, and some died of renal insufficiency following a superimposed nephrosclerosis. Considering the findings of the medical practitioner, such a course could hardly have been foreseen. It was, however, expected by the ophthalmologist, owing to the prognostic importance of marked crossing phenomena and the parallelism between the cerebral and the retinal vessels as well as the serious prognosis of the retinal condition.

Such apparent discrepancies prove conclusively the great value of ophthalmoscopic examination in cases such as I described in this article.

Clinical Notes

BILATERAL UVEITIS AND RETINAL PERIARTERITIS AS A FOCAL REACTION TO THE TUBERCULIN TEST

S. B. Muncaster, M.D., and H. E. Allen, M.D.
Washington, D. C.

A white woman aged 31, a school teacher, was seen on April 4, 1938, with acutely inflamed eyes, photophobia and markedly diminished vision. The county health physician was doing a routine tuberculin inoculation of all the school children; so the teacher decided to have the test done on herself. On March 21, almost two weeks before coming to our office, she was given 0.00002 mg. of purified protein derivative (Mulford). There was no reaction after forty-eight hours; on March 23 another injection was given, 0.005 mg. of the tuberculin being used. For both tests the injections were given intradermally in amounts of 0.1 cc. The reaction to the second injection was strongly positive, and after four hours both the first and the second test area became much inflamed. For the following ten days the patient felt tired and weak.

The left eye became red, watery, painful and sensitive to light. The right eye became similarly involved a few hours later. The patient went to an optometrist, who sent her to one of us (S. B. M.).

Her symptoms consisted of photophobia, lacrimation and congestion of the eyes. There was no diplopia, vertigo or coryza and no involvement of the sinuses.

Examination of the right eye showed profuse lacrimation, without mucus or pus. The lids were not swollen, but the conjunctiva was moderately hyperemic throughout. Around the cornea there was a characteristic deep, violet-tinged injection of the ciliary arteries. The cornea appeared hazy, the anterior chamber of moderate depth, the aqueous turbid, the pupil contracted and the markings of the brown iris indistinct. The eye was extremely sensitive to light. The tension was palpably soft and the vision 20/70. The pupillary reactions to light (direct and consensual) and to accommodation were present but were limited, owing to the contraction of the sphincter. The condition in the left eye was similar to that of the right except that the inflammatory condition was more pronounced and the vision only 20/200.

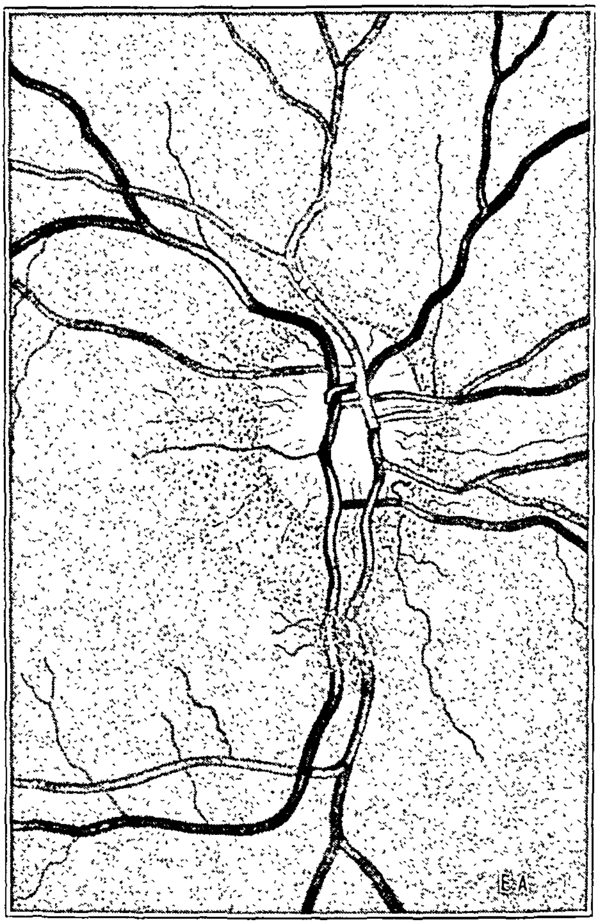
On ophthalmoscopic examination it was impossible to make out the fundus detail in either eye. The vitreous was cloudy after dilation. While the fundi were seen indistinctly, it could, nevertheless, be made out that there were no massive hemorrhages or exudates present.

Intraocular tension (Schiötz) in the right eye was 12 mm. and in the left eye 11 mm.

Examination with the slit lamp revealed thickened and edematous corneas. There were many wrinkles in Descemet's membrane and brown pigmented precipitates on the corneal endothelium in great profusion. The aqueous beam was strong, owing to floating cells and particles of pigment. The irides were muddy, the crypts contained inflammatory material, the reaction to light was sluggish, and the pupils were markedly contracted. The right iris seemed the more mobile of the two. There were some pigmented deposits on the left lens but not on the right.

There were a few small posterior synechiae in the right eye and a good many larger ones in the left.

A 3 per cent solution of atropine, epinephrine hydrochloride in a concentration of 1 : 1,000 and a 4 per cent solution of cocaine hydrochloride were instilled into the eyes, and hot compresses were applied for more than an hour. The right pupil dilated fairly readily, but there were more mature posterior synechiae in the left eye, which were slow in breaking up. However, both pupils were finally dilated, and the patient was sent to the Episcopal Hospital for thorough examination.



Picture of the patient's right fundus.

The only positive findings were a slight chronic tonsillitis and sinusitis, neither of which were thought by consultants to be responsible for the ocular condition. The Wassermann and Kahn reactions were negative. All examinations for tuberculosis, including roentgen examination, were negative except, of course, the tuberculin reaction.

When the patient first went to the hospital, 1 drop each of a 1 per cent solution of atropine and a 4 per cent solution of cocaine hydrochloride three times a day, rest in bed, dark glasses and hot compresses were ordered. As soon as both pupils were entirely dilated the drops of cocaine hydrochloride were discontinued and a drop of epinephrine hydrochloride in a concentration of 1 : 1,000 was substituted. The use of hot compresses and atropine were continued. For the first

six days in the hospital the only improvement noted was in the external appearance of the eyes, which were gradually clearing. After this, the media began to clear slowly, until on April 24 it was possible to make out small round spots of gray exudate resembling tubercles around the larger arteries near the disk in each fundus. By April 27 the media had cleared enough for an artist to make a sketch of the fundi.

The patient remained in the best of general health, and the media of the eyes cleared gradually, until on April 29 she was allowed to go home and remain in bed. The epinephrine hydrochloride was discontinued, but the use of atropine and hot compresses and the wearing of dark glasses were continued. At the time the patient left the hospital slit lamp examination showed a clear corneal epithelium, Bowman's membrane, corneal stroma and Descemet's membrane. On the endothelium there were fifteen or twenty rather large, scattered, hazy-edged, mutton-fat precipitates. There was no increase in the aqueous flare and no free floating cells in the anterior chamber. The pupil was dilated and did not react to light. There were no remaining posterior synechiae, although there were some pigmented specks on the anterior surfaces of the lenses. The same condition was found in each eye. The patient was seen in her home each week from the first of May until the middle of June, during which time her eyes gradually improved. On June 21 she appeared to be in the best of health. Corrected vision in each eye was 20/20. Only traces remained of the spots along the retinal arteries. There were, however, a few of these exudates that had not been absorbed, particularly one on the inferior branch of the retinal artery in the left eye in the disk. In all other respects the vessels were entirely normal. Their caliber was well maintained without spasm or constrictions, and there were no hemorrhages or exudates.

The color of the retina at the time of writing was just a little on the yellow side, but except for the beads of exudate around the larger arteries they were normal. The media were clear except for one spot on the back of the right cornea. The peripheral fields were normal.

FREQUENCY OF PHORIAS

Importance of Prism Correction

F. W. Dean, M.D., Council Bluffs, Ia.

During forty-five years of private practice in ophthalmology I have prescribed prisms incorporated in the lenses used for the correction of errors of refraction. The following record is not the record for all patients examined but only the record for those for whom glasses were prescribed. Each patient's record is kept in a separate space in the record books, so that the results can be easily inspected.

Having been taught that to correct an imbalance of the extrinsic muscles with prisms would aggravate the condition and that the amount of the imbalance would be increased, I began the use of prisms cautiously. Of the first 10,000 patients, 574 were given prisms for the correction of esophoria, 613 for the correction of exophoria and 288 for the correction of hyperphoria. More patients were found with esophoria than with orthophoria, and while they were comfortable with prisms base out, they were also comfortable with their refractive error corrected and without the prisms; therefore, the prism correction for

esophoria was discontinued. Because esophoria is found more often than orthophoria, a slight esophoria should be considered the normal condition.

Patients with exophoria and hyperphoria were much more comfortable with their phorias corrected. As time passed, the phorias neither increased nor decreased with the wearing of prisms; the amount remained constant. My conclusion was that the type of phoria for which prisms were given was not due to a lack of innervation but was an anatomic one. The reasons for this belief were stated in previous publications.[1]

The next 8,000 patients had their exophorias and hyperphorias corrected closely, 0.5 degree prisms being used in many cases. Of this group, 577 were given prisms for the correction of hyperphoria and 1,328 for the correction of exophoria. It was found that the patient with hyperphoria was much benefited with even the slight corrections. The patients with exophoria were comfortable with their corrections but got along well without the correction of a 0.5 degree error, so that at present the patients with exophoria have their errors corrected only if the phoria requires a 1 degree prism or more. In this second group of 8,000 patients for whom all the anatomic hyperphorias and exophorias were corrected, there were 1,905 who received prisms, or 1 in 4.45 of the patients for whom glasses were prescribed (22.5 per cent).

Of the next 3,000 patients (which brings the record to a recent date), 285 were prescribed prisms for the correction of hyperphoria and 223 for the correction of exophoria, making 508 patients, or 1 in every 6.02 (16.6 per cent) patients for whom glasses were prescribed.

In the literature stress is put on those phorias which are caused by failure of proper innervational impulses to the muscles or by lack of fusion sense or by whatever other cause might be ascribed. A glance at the foregoing figures show such phorias are not the most frequent cause of discomfort to the greatest number of patients.

There are three causes for which glasses should be prescribed: for protection, for vision and for comfort. Prisms are of no aid in glasses worn for protection against accident. They do not aid vision when added to the lenses given to correct refraction unless the phoria is so great that when the extrinsic muscles tire there is a resultant diplopia.

For comfort, the correction of phorias with prisms is essential. They change a condition of constant muscle strain with all the consequences to one of restful ease.

Judging from the examination of glasses which were prescribed in other offices, I conclude that errors of refraction have been carefully worked out, whether the examinations were made with the aid of a mydriatic or not, so that the strain on the intrinsic muscles is relieved. The extrinsic muscles are usually negelected. This accounts for a certain percentage of patients going from office to office seeking a relief which will give them comfort.

1. Dean, F. W.: Prisms: Should They Be Prescribed for Common Wear? Tr. Am. Acad. Ophth. **26**:108, 1921; Anatomic Phorias, Arch. Ophth. **15**:692 (April) 1936.

AN AID TOWARD CORRECTLY INSERTING CONTACT LENSES

Joseph I. Pascal, M.D., New York

The insertion of contact lenses having a uniform scleral curvature with a round corneal section set in the middle is fairly easy. The lens is placed on the rubber sucker without being oriented one way or the other, and the sucker, likewise without being oriented one way or the other, is approached to the eye and the lens inserted. But when the contact lens has different scleral curvatures in different directions and/or an oval corneal section centered or decentered in the scleral circumference, the process of insertion becomes considerably more difficult; for it is essential that such a lens be inserted so as to have its parts definitely related to parts of the eye. All lenses made to order from individual molds, as well as the newly introduced contact lenses with toric scleras, have to be placed on the eye in a definite position. There is an upper, lower, nasal and temporal side to the lens which has to be inserted so as to fit the corresponding part of the eye.

There is usually a fine etched line on the inner, nasal, side of the lens. The lens is inserted so that this line is directed toward the inner canthus. The procedure is as follows: The patient locates the etched line and places the lens on the sucker so that the line is horizontal and directed toward the inner canthus; then keeping the sucker in this position, he brings it up toward the eye and inserts the lens. Any rotation of the sucker during this process throws the lens out of position, but the displacement of the etched line on the lens would not be observed by either the patient or the physician, because the line, by its very nature, is thin and barely visible, especially if the patient has poor vision, which is frequently the case. Sometimes it is seen only when the lens is held close to the eye or up against a light. Once it is located and the lens, properly oriented, is set on the sucker, the line becomes invisible as soon as the patient looks away. The line, of course, is invisible to the physician, who can neither effectively aid nor check the patient in the procedure. When the physician inserts the lens, he has the same difficulty of more or less guessing the position of the etched line, once the lens has been placed and oriented on the sucker.

This difficulty can be largely overcome by the simple expedient of marking one side of the rubber sucker, preferably including half the top of the sucker, with a thin white line. A strip of white adhesive plaster from 1 to 2 mm. wide works well when placed on the side. The strip is conspicuous, can be seen from some distance by both the patient and the physician and acts as a guideline. To insert the lens, it is placed on the sucker with the etched line alined with the white strip. The patient does not have to watch or worry about the position of the etched line any more, as he is guided by the strip, which is a conspicuous landmark. The sucker is now held between the fingers so that the white strip is directed toward the inner canthus and is plainly visible in the hollow between the fingers. In this position the sucker is approached to the eye and the lens inserted.

When the lens is oval, with the horizontal diameter appreciably longer than the vertical diameter and the temporal section appreciably longer than the nasal section, it becomes much easier to set the lens properly on the sucker and to place the lens correctly on the eye. But even here the use of the white guideline is a help. On the other hand, it is desirable, even in ordering standard round contact lenses, to request that a thin etched line be placed at one edge. If the scleral portion has to be slightly ground off at some place where it exerts undue pressure, the place can be marked off with a pencil or located by the clock-face method. But this can be done only when there is an etched line on the lens to orient it properly both on and off the eye. The present development of contact lenses is in the direction of individually adapting even standard, nonmolded lenses; and the use of a guideline on the rubber sucker will materially aid in fitting these lenses.

Ophthalmologic Reviews

VERTICAL PRISM IMBALANCES IN BIFOCAL LENSES COEXISTENT WITH HYPERPHORIA

SIDNEY L. OLSHO, M.D.
PHILADELPHIA

In a prior paper [1] I advocated that the refractionist be conversant with the different types and makes of bifocal lenses and the individual optical attributes which qualify or disqualify any of them for a particular prescription.

This presentation supplements the preceding one. A few essentials will be reviewed.

REVIEW OF PREVIOUS ARTICLE

The eye is confronted by a prism not only when a prism is placed in front of it but also it must look through a lens at any eccentric point.

The modern bifocal lens is synthetic. The distance lens enters into the structure of the bifocal lens, and in reading, the line of sight passes unavoidably through an infracentric point.

In my previous work I assumed that the upper edge of a contemplated segment is 4 mm. below the optical center of the distance component and the reading level down an additional 4 mm., a natural reading point being arrived at which is a total of 8 mm. below the optical center of the distance component. This generally accepted depth for certain calculations can be modified when desired.

It will be found advantageous to consider the exact vertical prism values of the distance corrections at such two infracentric reading points. Precisely this information is useful when bifocal lenses are to be prescribed.

The vertical prism values are found by applying the Prentice principle, after estimating the vertical effective powers.

Vertical effective power was explained as follows:

Spherical lenses are 100 per cent vertically effective.

Cylinder lenses at axis 180 are 100 per cent vertically effective.

Cylinder lenses at axis 90 are not at all vertically effective.

Cylinder lenses at axis 45 and 135 are vertically effective 50 per cent.

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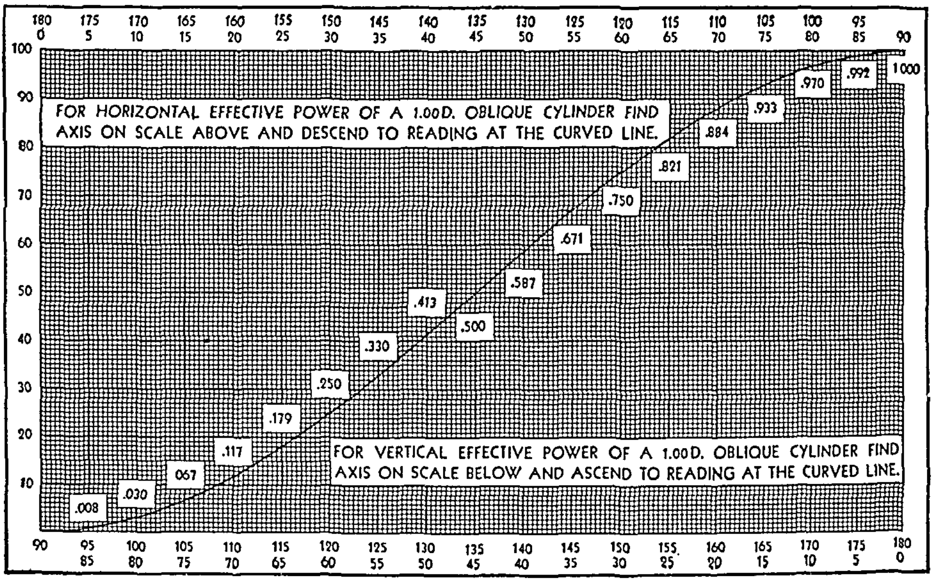
1. Olsho, S. L.: Vertical Prism Values in Commonly Used Bifocal Lenses, Arch. Ophth. **20**:95 (July) 1938.

Only a percentage of the power of, other oblique cylinders is vertically effective. This percentage depends on the axis, as can be seen on the accompanying graph.

The dioptric power multiplied by this percentage equals the vertical effective power of an oblique cylinder.

For spherocylinder lenses, to all of the spherical power is added the vertical effective power of the cylinder. The algebraic sum equals the total vertical effective power.



Graph used to estimate the net vertical effective power of any oblique cylinder. (Supplied by Bausch & Lomb Optical Company.)

Examples illustrating the estimation of the vertical effective power (V. E. P.) with aid of the graph follow:

(1)	+3.00 cyl., ax. 120	+3.00 × 0.25 = +0.75 V. E. P.
(2)	+2.00 cyl., ax. 150	+2.00 × 0.75 = +1.50 V. E. P.
(3)	—1.50 cyl., ax. 125	—1.50 × 0.33 = —0.50 V. E. P.
(4)	+3.00 sph. +2.50 cyl., ax. 75	+3.00 × 1.00 = +3.00
		+2.50 × 0.067 = +0.167
		+3.17 V. E. P.
(5)	—2.75 sph. +1.75 cyl., ax. 30	—2.75 × 1.00 = —2.75
		+1.75 × 0.75 = +1.31
		—1.44 V. E. P.

The vertical effective power of each main lens is multiplied by 0.8. The product is the vertical prism introduced by each main lens at the arbitrarily selected reading points.

Examples using the foregoing lens combinations follow:

(1) V. E. P. +0.75 × 0.8 = △ 0.6 B. U. at reading point
(2) V. E. P. +1.50 × 0.8 = △ 1.2 B. U. at reading point
(3) V. E. P. —0.50 × 0.8 = △ 0.4 B. D. at reading point
(4) V. E. P. +3.17 × 0.8 = △ 2.5 B. U. at reading point
(5) V. E. P. —1.44 × 0.8 = △ 1.2 B. D. at reading point

A brief summary in the aforementioned paper included the following points: The main, or carrier, lens is a component of the reading part of a modern bifocal lens. The main lens is centered for distance seeing. Near vision is obtained through a zone considerably below the optical center of the main lens. The main lens introduces a vertical prism to the composite reading part of the bifocal lens. The prism introduced to the reading part is base up or base down, depending on whether the total vertical effective power of the main lens is plus or minus. The vertical effective power of the main lens multiplied by the depth in centimeters of the reading point expresses in prism diopters the amount of prism introduced by the main, or carrier, lens to the composite reading part of a bifocal lens.

Pairs of lenses of approximately equal vertical effective powers and also segments were considered. It was pointed out that standard upright arch segments (according to the size of their circles of origin) contribute prism base down at the reading points. When the distance correction is convex it may happen that the base down prism of the addition is a perfect counterbalance to the base up prism already resident at the reading point. In such an occasional case optical centers are found in both parts of the bifocal lens.

It was shown that standard reversed arch or flat top segments neither add to nor subtract from the vertical prism already present at the reading points.

In modern bifocal lenses it is neither practicable nor necessary to eliminate all prisms at the reading points.

VERTICAL PRISM IMBALANCE

In lenses of unlike vertical effective powers there is a vertical prism imbalance at the reading points. It may be slight in amount, or there may be enough to make the bifocal lenses less efficient than they should be, uncomfortable or even unwearable.

Trouble involving costs, discomfort and reproach may be forestalled if the prescriber of bifocal lenses will ask himself: 1. In bifocal lenses made on this prescription will there be, within the natural reading zones, two corresponding points at which the vertical prism imbalance will be less than 1 prism diopter? If so, they may be tolerated. 2. Will the vertical prism imbalance at two natural reading points exceed 2 prism diopters? If so, prolonged fusion at the near point will be difficult, if it is possible.

Sometimes dissimilar segments are used to combat excessive vertical imbalance. At other times a compensating prism is introduced in one segment. The strategy varies. These expedients have their limitations and often have serious drawbacks, yet their employment would increase

if refractionists gave more general attention to vertical imbalance. These methods will not be discussed at this time. The need to resort to them is diminished by taking cognizance of the relation of hyperphoria. For example, a patient has right hyperphoria and the distance corrections are unequal. It follows that the lenses have a vertical prism imbalance at the reading points. If the prism which is there dominant is found to be appropriate for right hyperphoria it is good. If, however, the prism which is there dominant is found to be appropriate for left hyperphoria, it is bad. A brief discussion of hyperphoria seems pertinent, then further explanation.

HYPERPHORIA

It is of considerable value to give more than usual consideration to the measurement of hyperphoria when bifocal lenses are to be prescribed. The successful correction of hyperphoria is dependent on repeated careful measurement. The use of the appropriate vertical prisms, when indicated, tends to facilitate the unaided conquest of lateral imbalances.

The following recommendations may not be amiss:

The hyperphoria should be tested with a Maddox rod which gives the same reading after a rotation of 180 degrees.

The distance correction should be in the trial frame.

The patient must look through the centers of trial lenses, not above or below their centers, particularly if the two distance corrections are unequal.

If a mydriatic is used, the determination should be repeated after mydriasis.

If a mydriatic is not used, the determination should be repeated on other days.

The amount of hyperphoria should then be recorded with the Maddox rod before the right eye.

The rod should then be rotated 180 degrees and another reading made.

The amount of hyperphoria should then be recorded with the Maddox rod before the left eye. The rod should be rotated 180 degrees and another determination made.

All readings will not be alike.

The minimum prism which brings about a centering of the line on the distant light should be ascertained, repeated trials being made to establish if less prism is adequate.

Before a vertical prism is ordered, the patient's reaction to its presence should be tested, the correcting prism being alternately included and removed at brief intervals.

After these precautions all of the vertical prism necessary to produce the desired result should be prescribed.

CORRECTION FOR VERTICAL PRISM IMBALANCE COEXISTENT WITH HYPERPHORIA

In every instance in which bifocal lenses are contemplated a quick estimate of the vertical effective power of the two distance corrections should be made. If they are unequal, the accretion of vertical prism imbalance is extremely rapid in descent from the optical centers. An example in which this is obvious follows:

	Down 2 mm.	4 mm.	6 mm.	8 mm.	
R. +5.00 sph.	△ 1.0	△ 2.0	△ 3.0	△ 4.0	B. U.
L. +2.50 sph.	△ 0.5	△ 1.0	△ 1.5	△ 2.0	B. U.
Imbalance	△ 0.5	△ 1.0	△ 1.5	△ 2.0	

The excess prism is base up on the right eye at all levels.

Vertical prism imbalance at the reading points is not always so obvious. When cylinder axes are oblique, a mere glance at the formula may be deceptive. In the first of the following examples one would expect more vertical prism imbalance to be present. In the second example there is more prism disparity than a glance would lead one to suspect. The following abbreviations are used: V.E.P., vertical effective power; V.Pr., vertical prism; R.P., reading point, and B.U.R. and B.U.L., base up right and base up left.

Examples showing vertical prism imbalance at the reading points follow:

(1) R. —0.75 +1.75 cyl., ax. 15
L. —2.25 +3.00 cyl., ax. 165

V. E. P. R. = +0.88 V. Pr. is +0.88 × 0.8 = △ 0.70 B. U.
V. E. P. L. = +0.55 V. Pr. is +0.55 × 0.8 = △ 0.44 B. U.

Vertical prism imbalance at R. P. △ 0.26

(2) R. —3.00 —2.25 cyl., ax. 35
L. —2.75 —3.50 cyl., ax. 180

V. E. P. R. = —4.51 V. Pr. is —4.51 × 0.8 = △ 3.61 B. D.
V. E. P. L. = —6.25 V. Pr. is —6.25 × 0.8 = △ 5.00 B. D.

Vertical prism imbalance at R. P. △ 1.39

(3) R. +1.00 +2.75 cyl., ax. 180
L. +0.50 +1.25 cyl., ax. 60

V. E. P. R. = +3.75 V. Pr. is +3.75 × 0.8 = △ 3. B. U.
V. E. P. L. = +0.81 V. Pr. is +0.81 × 0.8 = △ 0.65 B. U.

Vertical prism imbalance at R. P. △ 2.35

Correction of even small amounts of hyperphoria is advisable if there is a dissimilarity of lenses resulting in unfavorable vertical prism imbalance in the reading zones. The correction of the hyperphoria can be omitted if with dissimilarity of lenses there is a favorable vertical imbalance in the reading zones. For example, the following is a commonplace refractive finding:

R. +1.75 +0.25 cyl., ax. 90
L. +1.00 +1.00 cyl., ax. 90 Addition +1.25
V. E. P. disparity +0.75 × 0.8 = V. Pr. disparity at R. P., △ 0.6 B. U. R.

For this patient repeated measurement disclosed a right hyperphoria of 0.75 prism diopter, for which the appropriate prism would be placed base up left. But in the lenses the vertical prism imbalance at the reading points is 0.6 prism diopter base up right. Unless a prism is prescribed with its base up left in the distance correction, there will be at the reading points an adverse load of 0.75 prism diopter of hyperphoria, plus 0.6 prism diopter of vertical prism imbalance, total 1.35 prism diopters. For this patient, therefore, I should make full correction of the hyperphoria. The residual prism would not be troublesome. The final prescription reads:

R. +1.75 +0.25 cyl., ax. 90
L. +1.00 +1.00 cyl., ax. 90 △ 0.75 B. U. Addition +1.25

If with the same refractive findings a left hyperphoria had been found for which in the lenses the vertical prism imbalance of 0.6 prism diopter base up right would be appropriate, my final prescription for bifocal lenses would be as first written without the prism.

The attempt is not made to eliminate all prisms nor to correct completely all hyperphoria in both distant and near use of bifocal lenses.

When a considerable amount of hyperphoria is present its correction should be *in full,* if there is a dissimilarity of the lenses and a dominance of unfavorable prism at their reading points.

The following refractive findings were found in such a case:

R. +0.50 +1.00 cyl., ax. 180
L. +2.00 +0.75 cyl., ax. 180 Addition +2.00

Repeated measurement disclosed a left hyperphoria of 2.5 prism diopters, for which the appropriate prism would be placed with base up right or base down left.

In the foregoing formula:

V. E. P. R. = +1.50 V. Pr. is +1.50 × 0.8 = △ 1.2 B. U.
V. E. P. L. = +2.75 V. Pr. is +2.75 × 0.8 = △ 2.2 B. U.

Vertical prism imbalance at R. P. = △ 1. B. U. L.

The lenses show at the reading points an adverse prism dominant.

A prescription therefore correcting preferably *all* of the left hyperphoria would read:

R. +0.50 +1.00 cyl., ax. 180 △ 2.5 B. U.
L. +2.00 +0.75 cyl., ax. 180 Addition +2.00

The correction for a considerable degree of hyperphoria should be judiciously moderated if with dissimilarity of lenses there is in the reading zones a favorable prism imbalance.

For example:

R. +4.50 +2.50 cyl., ax. 25
L. +2.75 +2.75 cyl., ax. 180 Addition +2.25

Repeated measurement disclosed left hyperphoria of 2.5 prism diopters, for which the appropriate prism would be placed with base up right.

V. E. P. R. +6.55 V. Pr. is $+6.55 \times 0.8 = \Delta\ 5.24$ B. U.
V. E. P. L. +5.50 V. Pr. is $+5.50 \times 0.8 = \Delta\ 4.40$ B. U.

Vertical prism imbalance at R. P. $= \Delta\ 0.84$ B. U. R.

At the reading points of the lenses the prism dominant is favorable to the hyperphoria. The correction for the hyperphoria is therefore moderated, and the prescription reads:

R. +4.50 +2.50 cyl., ax. 25 Δ 1.5 B. U. Addition +2.25
L. +2.75 +2.75 cyl., ax. 180

In the last prescription the hyperphoria is incompletely corrected in the distance portion of the bifocal lenses, but it is practically all corrected in the reading portion, viz:

Δ 1.5 B. U. + Δ 0.84 B. U. = Δ 2.34 B. U. R.

CONCLUSIONS

Consideration by the refractionist of the vertical effective powers of lenses and estimation of the vertical prism imbalances at the reading points provide the following advantages:

1. Excessive vertical prism imbalance in the reading zones is recognized prior to writing the prescription for bifocal lenses.

2. Purely optical expedients will not be resorted to which ignore the possible presence of hyperphoria.

3. Excessive vertical prism imbalance at the reading points may by a judicious correction of hyperphoria be brought within limits which can be tolerated.

4. Moderate amounts of hyperphoria may advantageously be corrected if in the lenses there is at the reading points a vertical prism imbalance unfavorable to the hyperphoria.

5. Moderate amounts of hyperphoria may advantageously be left uncorrected if in the lenses there is at the reading points a vertical prism imbalance favorable to the hyperphoria.

DISEASES OF THE CHOROID

MARTIN COHEN, M.D.
NEW YORK

Before the diseases of the choroid are reviewed, a few salient facts regarding the anatomic structure and the function of the choroid will be briefly mentioned.

The choroid lies between the retina and the sclera and extends from the optic disk to the ora serrata. It has a thickness of 0.3 mm. and is composed mainly of veins and arteries, principally the former. These vessels anastomose. The intravascular stroma is made up of elastic fibers, connective tissue fibers, chromatophores and nerves.

The following layers of the choroid can be distinguished from without inward: (*a*) the suprachoroidal space, consisting of elastic fibers and cells; (*b*) a layer of large blood vessels, the thickest layer of the choroid; (*c*) a layer of medium-sized blood vessels; (*d*) the choriocapillaris, a single layer of capillary blood vessels, and (*e*) the lamina vitrea, an elastic membrane. The choroidal vessels are branches of the ciliary vessels, which in turn are branches of the ophthalmic artery and vein.

The choroid is the vascular coat of the eyeball, and its function is mainly nutritional. Lesions of the choroid frequently extend to the retina, producing chorioretinal disease.

Choroidal diseases manifest themselves in an acute or chronic form. Acute choroiditis may be the result of local trauma or of a metastatic focus from a primary disease located elsewhere in the body, such as septic meningitis, pneumonia, septic endometritis and purulent sinus infections involving the orbit. These infections cause a septic choroiditis or a retinitis with opacities of the vitreous, simulating a glioma or metastatic choroiditis. The choroidal focus may become organized and eventually form a bony choroid. The primary focus should be treated early. Intense aggravation of the choroidal inflammation may lead to panophthalmitis and necessitate evisceration. The pathologic changes consist of an exudative inflammation of the choroid, which presents a nodular formation, associated with retinal edema, hemorrhages and con-

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This review is based on a course given at the Forty-Third Annual Session of the American Academy of Ophthalmology and Otolaryngology, Washington, D. C., Oct. 13-14, 1938.

gestion of the retinal veins. The choroidal focus is elevated and presents a rich infiltration of lymphocytes, leukocytes and sometimes bacteria.

Chronic choroiditis is usually the end result of an acute choroiditis, although the condition may be due to various systemic diseases of a chronic nature. The choroidal lesion consists of atrophic patches surrounded by lymphocytes, plasma cells and proliferated pigment. There is also a sclerosis of the choroidal vessels. These changes result in adhesions between the choroid and the retina (adhesive chorioretinitis). The treatment should be directed to the underlying cause of the chronic condition.

A brief review of choroidal diseases follows:

Syphilitic choroiditis occurs in both the congenital and the acquired form of syphilis and may involve the choroid alone, sparing the ciliary body and the iris. The fundus oculi may show isolated infiltrations or diffuse pigmentary changes. The lesion may become extensive, forming granulation tissue in the choroidal stroma; this granulation tissue consists of lymphocytes, plasma cells and mast cells. The granulation may be resorbed, leaving atrophic patches surrounded by proliferative pigment. The vasculature is frequently involved, showing sclerotic changes. The pigment epithelium is usually affected, owing to degenerative changes. In some cases there is an extrusion of degenerated pigment from the epithelial cells into the surrounding areas, causing disseminated foci, recognized as the pepper and salt fundus. Similar changes in the pigment occur in many chronic inflammatory diseases as well as in syphilis. In addition to the disseminated atrophic and pigmented foci, syphilitic choroiditis may be followed by extravasations into the vitreous, causing dustlike opacities. The lesions of the fundus are similar to those found in many chronic inflammations of the choroid, i. e., tuberculosis, sympathetic ophthalmia and other general or focal infections of long standing. For the differential diagnosis, a clinical history, a Wassermann test of the blood and of the spinal fluid and tuberculin tests are essential. Energetic antisyphilitic treatment should be instituted as early as possible in order to prevent late manifestations of syphilis. In cases of involvement of the optic nerve, arsenical preparations should be avoided and injections of a bismuth compound substituted.

In acute miliary tuberculosis, choroidal changes are rare unless the disease is complicated by meningitis, in which case a few yellowish gray, ill defined foci are scattered over the fundus in the retina. These foci are edematous retinal lesions occurring over the tubercles of the choroid. Pathologically, these tubercles are composed of giant cells, lymphocytes, epithelioid cells and sometimes tubercle bacilli.

The two forms of chronic tuberculous choroiditis are the conglomerate, or solitary, form and the disseminated form. Both types cause retinal involvement with adhesions (adhesive chorioretinitis). The solitary form occurs in children or in young adults and the disseminated form in older persons. The fundus shows lesions similar to those seen in syphilitic choroiditis or in other chronic forms of choroiditis. In the disseminated type, the choroid is thickened with infiltrations. Both forms show a slowly growing tumor-like area of granulation tissue, made up of giant cells, plasma cells and epithelioid cells. Tubercle bacilli are rarely found in the granulation tissue. Treatment consists of hygienic precautions, proper diet and injections of tuberculin and milk.

Sympathetic choroiditis is usually caused by ocular operations or local injuries and rarely by corneal ulcers or necrotic sarcomas. It consists of an infiltrating chronic inflammation of the choroid, involving primarily, as a rule, the anterior segment. The tissue at the site of focal inflammation consists of lymphocytes, polymorphonuclear cells, mast cells, nests of epithelioid cells and giant cells; the infiltrating mass resembles tubercles. These cellular elements may give rise to granulation tissue; resorption of the tissue is rare. The clinical signs are due to a plastic iridocyclitis in the injured eye. The clinical entity is characterized by its insidious course. The sympathizing eye is involved by a similar pathologic condition. The prognosis is generally grave; organization of the exudation occurs, with formation of scar tissue and other serious consequences. The pathogenesis is unknown. Treatment consists of early enucleation of the injured eye, which should be performed within two weeks if the usual medical remedies are ineffective and the clinical signs progress. Sympathetic ophthalmia may result in phthisis bulbi.

Myopic choroiditis is caused by a congenital stretching of the structures of the eyeball, including the vasculature, which leads to atrophic and pigmentary changes in the choroid and also to retinal hemorrhages and detachment of the retina. These lesions may exist even in cases of mild myopia and can be confined to one eye. The atrophic patches lack pigment epithelium, lamina vitrea, choriocapillaris and sometimes retina.

Traumatic choroiditis is the result of severe contusion or concussion of the eyeball and may be associated with rupture of the choroid. The rupture appears as an elliptic whitish area coursed over by retinal vessels and is generally located between the temporal margin of the disk and the macula.

Detachment of the choroid may follow an operation for cataract or iridectomy and is visible ophthalmoscopically as a blackish mass, which may be absorbed in the course of several weeks, leaving no ill effects.

Detachment of the choroid may occur after rupture of diseased choroidal vessels. Rupture of the vessels sometimes results in subchoroidal hemorrhages and subsequent formation of fibrous tissue. The prognosis of detachment of the choroid and retina following proliferating retinitis is unfavorable. Choroidal detachment may occur in cases of severe myopia, but this condition is rare.

In lymphatic and myelogenous leukemia or chloroma the choroidal vessels show collections of white blood cells, which cause a thickening of the choroid. The retinal veins are frequently engorged and tortuous. An increased leukocyte count of the blood confirms the diagnosis.

The choroid and retina are frequently involved in chronic diffuse glomerular nephritis. The walls of the choroidal vessels may be more affected than the walls of vessels in other parts of the body. As a rule, choroidal vascular sclerosis cannot be seen with the ophthalmoscope, owing to the density of the pigment epithelium. The choroidal lesions in chronic diffuse nephritis include edema, hemorrhages, serofibrinous exudates and hyperplastic sclerosis of the arterioles.

The choroidal lesion in diabetes is caused by glycogenic deposits in the choroid and the retina and also by vascular sclerosis. The retinal changes consist of lipoid depositis, seen as glistening white, sharply defined foci in the retina, and of small circular hemorrhages. The optic disk usually appears normal. In hyperlipemia occurring in cases of severe diabetes (lipaemia retinalis) the increased fat content of the blood can be recognized by a similar milky appearance of the retinal veins and arteries. The choroidal vessels also show an increase in lipoid material.

Lesions of the vascular walls are frequently seen in the choroid in essential hypertension, chronic glomerular nephritis and arteriosclerosis, especially if the retinal pigment is partially deficient or degenerated. The lesion consists of a hyperplastic intimal thickening with regressive changes, such as hyaline and lipoid degeneration, causing encroachment on the lumen of the vessels.

Drusen, or excrescences of the lamina vitrea, are probably the result of lesions of the vascular walls occurring in the aged. These excrescences are chronic inflammatory foci which may become fibrous and caseous and irritate the outer retinal layers. Drusen appear as fine white dots which are scattered over the fundus area. In senility the fundus may also show yellowish dots surrounded by fine pigment in the macular area and calcareous deposits. The walls of the choroidal vessels may show a fibrous thickening without encroachment on the lumen.

The choroidal lesion in leprosy is produced by an extension of the anterior uveal lesion. The ciliary nerves are affected by this lesion. Lepra bacilli have been isolated in the choroid.

A choroidal tumor may be either primary or secondary. If it is primary, it is a sarcoma, generally a melanosarcoma, consisting of pigmented spindle or round cells and sometimes of both. Metastases may occur in other organs. Hemorrhages and necrosis often occur within local areas of the tumor, owing to vascular lesions. The melanosarcoma is a malignant growth, and the involved eye should be enucleated early to prevent metastasis. If it is not enucleated, the eyeball will shrink and show signs of iridocyclitis. In cases in which extension has occurred into the orbital tissue, there is a possibility of metastasis even after enucleation.

Melanoma of the choroid is rare. Ophthalmoscopically, it appears as a flat, slate-colored pigmentation of the choroid. If over a long period there are increasing visual defects and evidences of an enlargement of the growth (as evidenced in photographs of the fundus), a melanoma should be considered.

If the growth is secondary it is a carcinoma, which usually metastasizes from an adenocarcinoma of the breast and also of the lungs, prostate and other organs. Pathologically, the intraocular carcinoma shows the same morphologic structure as the primary growth. The ischemia causes necrosis of the tumor mass. The treatment for the secondary growths is necessarily symptomatic.

An intraocular tumor is recognized ophthalmoscopically by its elevation into the vitreous, its sharp outline, the straight course of the retinal vessels over the surface of the tumor, a superficial pigmentation over the growth and a fresh retinal detachment at the border of the growth. Scleral transillumination and increased intraocular tension may be of added value in the diagnosis.

News and Notes

Edited by W. L. Benedict

GENERAL NEWS

Collected Papers of Dr. John M. Wheeler.—The staff of the Institute of Ophthalmology of the Columbia-Presbyterian Medical Center, New York, will publish a memorial volume of "The Collected Papers of Dr. John M. Wheeler," the majority dealing with ophthalmic and plastic surgery. This book will be distributed in the near future.

Any one desiring to reserve one or more copies at the cost price of $4 per copy (postage prepaid) should send a check to the Library Committee, Institute of Ophthalmology, 635 West One Hundred and Sixty-Fifth Street, New York.

UNIVERSITY NEWS

Graduate Course at the New York Eye and Ear Infirmary.—The School of Ophthalmology and Otolaryngology of the New York Eye and Ear Infirmary offers its annual postgraduate course from March 27 to April 1. The course will consist of lectures during the morning hours and case presentations and operations during the afternoon. Each day the matriculates will be the guests of the school at luncheon, when the morning lecturers will answer questions of the students. The number of matriculates will be limited. The fee for the course is $50, of which $10 must accompany the application. For information, address the School of Ophthalmology and Otolaryngology, New York Eye and Ear Infirmary, Thirteenth Street and Second Avenue.

SOCIETY NEWS

Congress on Ophthalmology.—The International Association for the Prevention of Blindness will meet in London, England, on April 19, preceding the annual congress of the Ophthalmological Society of the United Kingdom. Use of the Credé method for the prevention of ophthalmia neonatorum will be the subject for discussion at the international meeting. The speakers will include Dr. Conrad Berens, New York; Dr. Arthur H. H. Sinclair, Edinburgh, Scotland; Prof. F. Terrien, Paris, France; Prof. V. Szily, Munich, Germany; Prof. Luigi Maggiore, Genoa, Italy; Prof. Vasquez Barriere, Montevideo, Uruguay; Dr. Rowland P. Wilson, Cairo, Egypt, and Dr. John D. M. Cardell, London, England. This association may be addressed at 66 Boulevard Saint-Michel, Paris VI. Also on April 19 the International Organization Against Trachoma will hold a meeting to discuss the incidence and types of trachoma in various parts of the world. The speakers will be Dr. Harry S. Gradle, Chicago; Dr. Francis J. Lavery, Dublin, Ireland, and Mr. Arnold Sorsby, London, England. The address of this organization is 33 Welbeck Street, London, W. 1.

Obituaries

B. R. KENNON, M.D.
1871–1938

Dr. Beverley Randolph Kennon, of Norfolk, Va., died at his home on Dec. 27, 1938.

He was born in Powhatan County, Virginia, on Oct. 15, 1871, the son of William Upshur and Bessie Gilliam Kennon. His early education was received there.

In 1891 he graduated from the Virginia Polytechnic Institute, with the degree of Bachelor of Science, and in 1893 he received the degree of Doctor of Medicine from the University of Virginia.

From 1894 to 1896 Dr. Kennon did psychiatric work in the state of New York. He was a member of the house staff of the New York Eye and Ear Infirmary from 1897 to 1899.

During his medical career, Dr. Kennon served as an attending ophthalmologist and otologist at the Norfolk General Hospital. He was past president of the following organizations: the Norfolk County Medical Society, the Seaboard Medical Society, the Virginia Otolaryngological and Ophthalmological Society and the New York Eye and Ear Infirmary Alumni Society. He was vice president of the American Laryngological, Rhinological and Otological Society. He was also a past president of the Society of the Cincinnati of the State of Virginia. During the World War, he had the rank of major in the United States Army and served in France.

He married Ruth Alexander Ferrbee, of Norfolk. They had two children, a daughter, Ruth Ferrbee Kennon, and a son, Dr. Beverley Randolph Kennon III.

Dr. Kennon was an excellent physician and was highly regarded by his colleagues and friends.

CLYDE E. MCDANNALD, M.D.

ESTEBAN CAMPODONICO, M.D.

1867—1938

The many friends of Dr. Esteban Campodonico, of Lima, Peru, regret his death, on Oct. 23, 1938. When en route for Peru in September he contracted pneumonia and was transferred to the Gorgas Hospital, at Ancon, where he died two days later.

Dr. Campodonico was a student and a linguist, speaking five languages fluently. He graduated with honors from the University of Lima, Peru, and from the University of Bologna, Italy, receiving his medical degree in 1896. In 1898 he began the study of ophthalmology, and in 1907 he was made chief of the Italian Hospital at Lima. In 1909 he studied at the University of Vienna. On Jan. 5, 1909, he was made a Knight of the Order of the Crown of Italy; later he was raised to the rank of Official Knight, and on Oct. 22, 1922, by royal decree, he was made Commander Knight. In 1928 he was appointed by the Pope a Knight of the Order of San Gregorio Magno. In 1932 he was appointed Knight of the Order of San Marizio and Lazaro for his contribution to ophthalmology and radiology.

Dr. Campodonico was a member of the leading medical societies of Peru and represented his country in many international meetings. He was a member of the American College of Surgeons.

He contributed largely to the literature on ophthalmology and radiology, being a pioneer in the latter subject in his country. Much of his time was given to charity.

Dr. Campodonico was an enthusiast in aviation and frequently took a plane to the United States.

He was married to Miss Ethel Graff, of Oakland, Calif., in September 1938.

I was privileged in knowing Dr. Campodonico for the past twenty years and feel a deep personal loss in his death.

J. W. White, M.D.

Abstracts from Current Literature

Edited by Dr. William Zentmayer

Biochemistry

Action of Ciliary Body Extract on Tissue Oxidation. D. Michail and P. Vancea, Compt. rend. Soc. de biol. 129: 15, 1938.

Ciliary body extract retards the decoloration of methylene blue by inhibiting the power of tissue oxidases. This inhibitory action is annulled, however, by epinephrine.

J. E. Lebensohn.

Color Sense

Methods of Testing for Colour Vision and Theoretical Deductions from Observations on Colour Vision. H. E. Roaf, Brit. M. J. 2: 440 (Aug. 27) 1938.

This is a technical paper which does not lend itself to abstracting in its entirety.

The author notes that the Ishihara plates are probably the best for testing color vision. He states that a normal person, when using these plates, classes yellow-red or vermilion and blue-red or carmine together but distinguishes them from yellow-green and blue-green.

The usual type of defective color vision is failure to distinguish red, yellow and green, that is, red-green confusion, but no failure to distinguish blue from green. The normal person and the hypochromat distinguish blue from not-blue. The normal person distinguishes red from not-red. That which is neither red nor blue is called green. The hypochromat does not distinguish red from not-red, and anything which is not blue he tends to call yellow, as that is the brightest part in the not-blue region of the spectrum.

W. F. Duggan.

Cornea and Sclera

Icteric Discoloration of the Cornea. J. Boeck, Arch. f. Ophth. 138: 790 (July) 1938.

In 13 of 18 cases of jaundice the author discovered by slit lamp examination a yellowish green discoloration of a portion of the cornea which, in the optical section, was wedge shaped, the base of the wedge lying at the limbus and the apex lying in the deepest layers of the cornea, about halfway between the limbus and the center of the cornea. The intensity of the discoloration was directly dependent on the degree of the bilirubinemia and the duration of the jaundice but also tended to be more pronounced in cases of jaundice due to disease of the parenchyma of the liver and in young persons. In stained or unstained sections of the corneas of persons who died of jaundice the discoloration was not visible. If the patient recovered from the disease which caused the jaundice, the discoloration of the cornea disappeared pari

passu with the disappearance of the bile pigments from the skin. The author confirmed the absence of bile pigment from the tear fluid in cases of jaundice. Whether or not the aqueous contains bilirubin in cases of jaundice in man is not known. The fact that only certain portions of the cornea become stained allows certain speculations with regard to the metabolism of the cornea. The unstained portions seem to be less dependent on the surrounding sclera and its vessels.

P. C. KRONFELD.

Experimental Pathology

EXPERIMENTAL "EXOPHTHALMOS" IN DOGS. H. E. ESSEX and W. C. CORWIN, Am. J. Ophth. **20:** 153 (Feb.) 1937.

Essex and Corwin describe an experimental exophthalmometer, and after experiments on dogs make the following observations:

"This series of experiments indicates that the so-called exophthalmos reported by previous workers as resulting from sympathomimetic drugs is more correctly explained as advancement of the eye to its normal position from a condition of enophthalmos caused by the anesthetic. Chloralose, the anesthetic commonly used in previous investigations, gave the most profound enophthalmos we have seen of any of the intravenous anesthetics used.

"Measurements on the position of the eye made by the use of a simple instrument have shown that: anesthetics such as pentothal sodium, sodium amytal, pentobarbital sodium, and chloralose, administered intravenously, cause marked enophthalmos; surgical ether anesthesia produces slight exophthalmos; the ephedrine, tyramine, and electrical stimulation of the vagosympathetic nerve of dogs anesthetized with certain intravenous anesthetics cause the eye to advance almost to the position seen when the animal is in a conscious state.

"In the unanesthetized animal, ephedrine and tyramine in appropriate doses do not produce an exophthalmos worthy of note.

"Tyramine causes the muscular cone of the eye of a dog under anesthesia with pentobarbital sodium to constrict in an identical manner and for a period comparable to that resulting from electrical stimulation of the cut vagosympathetic nerve.

"Repeated daily doses of tyramine do not cause exophthalmos in normal or experimentally thyrotoxic dogs."

W. S. REESE.

THE PRODUCTION AND CURE OF OCULAR DISTURBANCES IN ADULT ALBINO RATS BY ADJUSTMENT OF VITAMIN A. A. M. YUDKIN, A. U. ORTEN and A. H. SMITH, Am. J. Ophth. **20:** 1115 (Nov.) 1937.

The authors discuss the work on vitamin A in relation to the age of the experimental animal and conclude that when the entire source of vitamin A is removed an adult animal which has little or no store of vitamin A is fully as susceptible to the deficiency as is a young rat with its low reserve of the vitamin. The results of vitamin A deficiency as shown by the tissues in experimental animals and also the repair that follows its addition to the diet are enumerated. Sixteen patients

with keratitis were observed who were apparently benefited by the use of cod liver oil and large quantities of vitamin B complex.

W. S. Reese.

Experimental Studies of the Intraocular Tolerance of Inoxidizable Industrial Steel and Alloys. M.-A. Dollfus and I. Borsotti, Bull. Soc. d'opht. de Paris **50:** 188 (May) 1938.

The incentive for this investigation lies in the fact that industry is using more and more inoxidizable steel and alloys which are feebly magnetic. A comparison was made in the rabbit's eye between ordinary magnetic steel and these other compositions in order to ascertain degrees of tolerance.

Two methods of introduction of the metal into the eye were used: (1) anteriorly after paracentesis of the cornea with peripheral iridectomy and (2) posteriorly through a conjunctival scleral opening near the equator. A general anesthetic was used, and the position of the foreign body was verified by the ophthalmoscope. Conclusions are as follows:

1. Fragments of *inoxidizable metal are less easily drawn by the* magnet.

2. The eye is remarkably tolerant of such inoxidizable metals, there being a minimum reaction even after a period of six weeks.

3. Contrariwise, ordinary steel and nonmagnetic nickel-copper are poorly tolerated. Reactions are immediate and progressive.

4. Histologic studies reveal a siderosis when ordinary steel remains in the eye, while the inoxidizable metals show no siderosis after periods as long as two weeks.

L. L. Mayer.

General

Contact Dermatitis from "Horn-Rimmed" Spectacles. H. S. Berkoff, Arch. Dermat. & Syph. **38:** 746 (Nov.) 1938.

Dermatitis from "horn-rimmed" spectacles is apparently rare. In the reported instance irritation developed after the frames had been worn ten days. The patient was not sensitive to the dye (nigrosine) nor to cellulose acetate, cellulose nitrate, acetic acid or gum camphor. However, the reactions to tricresyl and triphenyl phosphates, softening substances usually incorporated in such frames, were such as to implicate them as the probable causative factors.

J. E. Lebensohn.

General Diseases

Iritis in Undulant Fever. M. H. Viallefont, Arch. Soc. d. sc. méd. et biol. de Montpellier **19:** 77, 1938.

Iritis developed in a patient with undulant fever of nine months' duration; atropine gave no relief. However, a series of five injections of neoarsphenamine terminated both the iritis and the fever.

J. E. Lebensohn.

OCULAR MANIFESTATIONS OF MALIGNANT ENDOCARDITIS. P. BONNET and G. BONAMOUR, J. de méd. de Lyon 19: 191 (March 20) 1938.

Bonnet and Bonamour state that malignant endocarditis of slow evolution, appearing as a preliminary septicemia grafted on old valvular lesions, can manifest itself in exceptional cases by a sudden dramatic visual disturbance, a unilateral amaurosis caused by embolism of the central artery of the retina. During the evolution of malignant endocarditis, when attention is not called to the eye by any functional trouble the systematic examination of the fundus reveals in 40 per cent of the cases retinal changes which are of great value in the diagnosis of the disease. The retinitis of Roth—clearly outlined hemorrhages, roundish or oval and of navicular form, with white centers and white spots without hemorrhage—seemingly represents the most characteristic change in the chronic form of malignant endocarditis of slow evolution. These hemorrhages appear rapidly in several hours, becoming obliterated and reappearing in other places. They are probably the foci of necrosis limited to the retina at the level of the microbic colonies disseminated by the blood stream in the layer of the nerve fibers. More massive hemorrhages with white central foci may also be observed in front of the vessels in a difuse edematous process of the retina. It is not exceptional to observe forms which by their association with the changes in the unveal tract and their inflammatory character permit septic retinitis to be considered as an attenuated form of metastatic ophthalmia.

The observation of retinal changes remains, although less than was formerly asserted by Litten, a sign of an unfavorable prognosis, quite often the indication of an early fatal outcome.

W. ZENTMAYER. [J. A. M. A.]

Glaucoma

CYCLODIALYSIS WITH POSTERIOR SCLERECTOMY. S. N. IVANOV, Vestnik oftal. 11: 519, 1937.

Ivanov combines cyclodialysis after the method of Heine with posterior sclerectomy in order to avoid reactive hypertonia, which is observed when cyclodialysis alone is performed. No filtering scar is formed here because the defect in the sclera is filled with the material supplied by the suprachoroidal area and the sclera.

The technic of the operation is described. A triangular piece is incised from the sclera with its base toward the limbus. The cyclodialysis is done in the usual manner. Fifteen patients with advanced glaucoma with markedly constricted visual fields were operated on. Hypotonia developed in 7 patients after the operation, the tension remained normal in 5, and in 1 patient the tension increased. The vision improved in 4 patients and was impaired in 3, while in the others it remained unchanged. There was marked narrowing of the visual fields in 10 patients, which did not improve after the operation. Ivanov believes that this combined operative intervention has the following advantages: Posterior sclerectomy leads to no postoperative complications. This absence of hypertonia makes it possible to use mydriatics if an irritation of the ciliary body develops. The hole in the sclera makes the out-

flow of the blood easy. Hypotonia following the operation aids the gaping of the supraciliary space, and possibly a contraction of the ciliary muscle in the detached ciliary body develops.

O. SITCHEVSKA.

Lacrimal Apparatus

SYMMETRIC LYMPHOMAS OF THE LACRIMAL GLANDS. D. MICHAIL, Ann. d'ocul. **175:** 565 (Aug.) 1938.

Diseases of the lacrimal gland are rare and often present more or less obscure problems. Symmetric disease of the glands has been much discussed since the attention of oculists was called to this condition by Mikulicz in 1892, who reported on a symptom complex characterized by symmetric swelling of the lacrimal and salivary glands, which has since been known as Mikulicz' disease. Since that time much research has been done on the etiology of the condition.

The result of these researches may be divided into three groups, in the first one of which is placed tuberculosis as the etiologic factor. In the second group syphilis is put forward as the cause, and in the third, the disease is attributed to a hyperplasia of the lymphoid tissue without designation of the precise nature of this hyperplasia.

Two cases are described in detail. The first was that of a patient 30 years of age, who one year after typhoid presented a swelling of inflammatory aspect, which began at the level of the left lacrimal gland and later developed on the right side in a similar manner. These enlargements in the course of their development went through periods of exacerbation and thinning of the inflammatory parts on to progressive glandular hypertrophy, which became troublesome on account of the diplopia which it caused. In the second case, that of a young man aged 21, during three months symmetric lacrimal tumors developed accompanied by exophthalmos. Removal of the tumors was followed two months later by a recurrence of one of the growths and a bilateral infiltrating sclerokeratoconjunctival lesion. Seven illustrations and a bibliography accompany the article.

S. H. MCKEE.

OPERATION FOR EVERSION OF THE LOWER LACRIMAL PUNCTUM. P. E. TIKHOMIROV, Vestnik oftal. **11:** 216, 1937.

Tikhomirov found that the various operative methods for correction of epiphora due to the eversion of the lower punctum were unsatisfactory. He reports his own operative procedure, which he illustrates by a drawing.

A rhomboid-shaped flap of the conjunctiva is incised at the inner angle. The apex of the flap is toward the fornix; the lower part, which continues into a long strip of the conjunctiva, is directed toward the temple. The strip is about 25 mm. long and 2.5 mm. wide, and the rhomb is about 5 mm. wide. From three to four sutures are applied. This procedure shortens the lid and turns it toward the eyeball. The patients reported relief from the epiphora in from four to five days after the sutures were removed. The operation is simple and the patient may be ambulatory.

O. SITCHEVSKA.

Lens

Cataract of Myotonic Dystrophy. T. A. Vos, Ann. d'ocul. **175:** 641 (Sept.) 1938.

After studying myotonic dystrophy for four years, Vos has succeeded in assembling a sufficient number of cases of this strange malady to proceed with an analysis and classification. In the course of the study the condition of the eyes was especially noted. In a preliminary article published in December 1936 he reported 28 cases, and since that time he has collected 11 more. Of the 39 patients, 15 were men and 24 women. Their respective ages, the symptoms complained of and the condition found are given.

The second section of the article deals with the family history of these patients and is presented in considerable detail. Regarding the ophthalmoscopic examination, the author states that all patients with myotonic dystrophy have opacities of the lens of the character described by Vogt. The Vogt type is not necessarily related to the age of the lens. It develops in young persons as well as in the aged. Other ophthalmologic symptoms, such as ptosis, ectropion and dystrophy of the cornea, are more or less as frequent as opacities of the lens.

There are 15 illustrations, including photographs of patients, genealogic charts and tables.

S. H. McKee.

Experimental Studies on Therapy with Lens Antigen. D. Rapisarda, Ann. di ottal. e clin. ocul. **66:** 183 (March) 1938.

The author injected intravenously into rabbits emulsions of beef lens and rabbit lens in increasing doses. Severe reactions were produced by the larger doses, even in animals which had received no previous injections. The author considers that such reactions are due to a toxic substance present in the lens and not to anaphylaxis. The animals were divided into two groups, according to whether rabbit lens or beef lens antigen was employed for immunization. All the animals were subjected to discission of the lens, which was as complete as possible. Each group was subdivided into three subgroups, one of which was treated with lens antigen sometimes before discission, one by injections begun at the time of discission and one twenty days after discission. The serum of the animals was tested for precipitins with extracts of beef, human and rabbit lenses. An equal number of control animals which received no lens antigen was observed after discission, the animals being paired according to similarity in the immediate result of discission.

No influence on the absorption of cortical material was observed in any of the groups treated with either beef or rabbit antigen. No effect on the absorption of cortex in one eye was observed after discission of the opposite lens. No reactive phenomena were observed which could be attributed to sensitization produced by the injections. Twenty-four rabbits were borne by animals in the course of immunization, but none of these showed any lesion of the crystalline lens.

The results of precipitin tests in all the groups were uniformly negative.

S. R. Gifford.

THE AMINO ACIDS OF THE LENS IN PARATHYROIDECTOMIZED RABBITS. A. MILANO, Ann. di ottal. e clin. ocul. 66: 393 (May) 1938.

The author estimated the total amino nitrogen in 12 normal lenses of rabbits and in 20 lenses of rabbits after parathyroidectomy. There was a constant increase in weight of the lenses amounting on the average to 21 per cent. While the actual percentage of amino nitrogen was no greater after operation when allowance was made for the increased volume of the lens due to imbibition of water, the amount of amino-nitrogen showed a definite increase, being 48 mg. per hundred cubic centimeters compared with 40.5 mg. in normal animals. The amount was greater, as a rule, in lenses with marked opacities as compared with that in 4 lenses which remained clear after operation. The author believes that his findings indicate that splitting of the lenticular proteins is more important in the formation of opacities than slowing of the lens metabolism.

S. R. GIFFORD.

Neurology

PERIARTERITIS NODOSA: A CLINICOPATHOLOGIC STUDY WITH SPECIAL REFERENCE TO THE NERVOUS SYSTEM. JAMES W. KERNOHAN and HENRY W. WOLTMAN, Arch. Neurol. & Psychiat. 39: 655 (April) 1938.

Five cases of periarteritis nodosa are presented with careful clinical and pathologic studies. A review of the literature revealed only 2 previous reports in which microscopic changes in the eyes were described. In 3 cases of the present series the peripheral nervous system was widely degenerated; in 1 case the hair was involved and in the other, the choroid of the eye. In the last case the eyes showed clinical signs of hypertensive retinitis with separation of the retina simulating the picture seen in eclampsia.

The arterioles of the retina showed thickening of the media, which was the result of hypertension. In approximately 20 per cent of the arterioles of the choroid there existed hyalin-like necrosis of the media. This tissue was fragmented and clumped into small masses and simulated the changes seen in amyloid disease. It stained bright pink with eosin and did not contain any nuclei. No inflammatory reaction had yet occurred in the media. There was no proliferation of the intima, and although all the arteries in which necrosis of the media had occurred were widely dilated and irregular in outline, one could not be certain that aneurysms were beginning to form. Choroidal arterioles normally have little adventitia, but around the arterioles in which necrosis of the media had occurred slight proliferation of adventitial connective tissue was observed. As a rule there was no periarteritis, but around one or two arterioles small collections of lymphocytes were seen. However, there were no polymorphonuclear leukocytes or foreign body giant cells.

R. IRVINE.

MECHANISM OF MIGRAINE HEADACHE AND ACTION OF ERGOTAMINE TARTRATE. J. R. GRAHAM and H. G. WOLFF, Arch. Neurol. & Psychiat. 39: 737 (April) 1938.

Data produced in the paper lend support to the postulate that the head pain of the migraine attack is produced by the distention of cranial arteries and that termination of the headache by the administration of ergotamine tartrate is due to the capacity of this agent to constrict these cranial arteries and thus reduce the amplitude of their pulsations.

The dilatation of the temporal vessels was noted to lessen following injection of ergotamine tartrate, which stopped the attack of migraine. Similar observations followed pressure on the common carotid artery. The migraine headache, having been controlled by ergotamine tartrate, could be started again by injections of histamine, resulting in a recurrence of pulsatile dilatation of the temporal vessels.

Boring pain back of the eye during an attack of migraine is common. This pain is not controlled by pressure over the temporal or occipital arteries. The ophthalmic artery and its branches, the frontal and supraorbital arteries, the external carotid artery near its source and the middle meningeal artery are suggested as possible sites of origin of pain back of the eye.

R. IRVINE.

SYNDROME OF CLAUDE-BERNARD-HORNER PROVOKED BY A MASSIVE RETROBULBAR INJECTION OF ACETYLCHOLINE. P. MICHAND, Bull. Soc. d'opht. de Paris 50: 138 (March) 1938.

A woman aged 46 had a sudden obliteration of the central vein of the retina of the right eye with a decrease in vision to 1/50. Five centigrams of acetylcholine was given immediately. The results were poor, and three days later 6 cg. was given. There was slight amelioration of the vision, with paralysis of the superior and external rectus muscles. Later complete paralysis of the cervical sympathetic nerves ensued and has remained.

The extraocular muscles recovered in about one month's time. The visual acuity returned to 0.8. The possibility that the cervical sympathetic nerves may be influenced by large dosage of such a drug must be considered.

L. L. MAYER.

CHIASMIC SYNDROME OF A CRANIOPHARYNGIOMA IN A SYPHILITIC SUBJECT. A. RUBINO, Riv. oto-neuro-oftal. 14: 484 (Sept.-Oct.) 1937.

Rubino reviews 1 case in which a craniopharyngioma produced a chiasmic syndrome. The diagnosis was confused by the presence of a syphilitic infection. On the basis of clinical findings alone, a syphilitic neuritis would have explained the symptoms, but roentgenographic examination revealed the presence of an expansible neoplasm beneath the chiasm.

Rubino advises an early exploratory craniotomy in cases in which the diagnosis is doubtful for the purpose of finding the true pathologic process and to obtain improvement of vision that usually follows such surgical intervention.

F. P. GUIDA.

Ocular Muscles

Suppression of Vision in Squint and Its Association with Retinal Correspondence and Amblyopia. T. ÀB. Travers, Brit. J. Ophth. 22: 577 (Oct.) 1938.

A color test for suppression is described. It demonstrates a functional scotoma in the deviating eye of squinters. This scotoma surrounds a point which lies in the same visual direction as the fixing macula. It is present in whichever eye is squinting at any particular moment. When that eye holds the attention, the scotoma ceases to exist and is transferred to the other eye. Suppression is most marked at the center and fades toward its periphery. A new method of mapping a suppression field is described. The tests show that a regional suppression in one eye is produced by the act of fixation in the other eye. Abnormal correspondence prevents the development of amblyopia. Worth's classic grades of binocular vision are shown to be inadequate. To determine if correspondence is normal or abnormal, one must know two values, the angle of squint and the "angle of binocular projection." It is suggested that tests such as the synotophore method or the mirror-screen, which measures the binocular projection lines of the two maculas, are the best methods of diagnosing abnormal correspondence and measuring the angle of anomaly. It is suggested that the various grades of the first stage of binocular vision described indicate only different degrees of suppression.

W. Zentmayer.

Concerning Paralyses of Supranuclear Origin: Attempt at Classification. G. E. Jayle, Bull. Soc. d'opht. de Paris 50: 144 (March) 1938.

The author would differentiate between two types of permanent deviation of the eye, one in which deviation is characterized by inability to move the eye in any other direction, giving the appearance of a paralysis, and the other in which there is a fixed regard with slight movements in other directions, as though a spasm were present. In the latter type one thinks of a contracture. An example of this type is that found in the postparkinson deviations in which scopolamine is of benefit in relieving the contracture. The classic division as made by Alajouanine and Thurel and concurred in by Morax consists of two groups: (1) complete paralysis, which is noted at the time of voluntary or automatic movement in the direction of regard, and (2) dissociated voluntary paralysis acting on voluntary movement.

Jayle's classification is as follows: (1) isolated paralysis of movement, as in optokinetic nystagmus, the paralysis existing in only one direction; (2) a paralysis of the supernuclear origin limited to one eye only; (3) paralysis in the classic syndrome of Foville; (4) paralysis associated with oculovestibular conditions, and (5) complete binocular paralysis with nystagmus.

L. L. Mayer.

Orbit, Eyeball and Accessory Sinuses

Immediate Effects on Ocular Tonus of the Retrobulbar Injections of Procaine Hydrochloride with Epinephrine. C. Dejean, R. Guignot and P. Artières, Bull. Soc. d'opht. de Paris **50:** 198 (May) 1938.

All measurements were determined on patients previous to intracapsular extraction for cataract to ascertain (1) the value of the hypotonia obtained (2) the rapidity of its appearance, (3) its duration and (4) its variations.

All the patients received a subconjunctival injection of epinephrine hydrochloride (1:1,000) to establish persistent mydriasis. The ocular tension was then measured with the Schiötz tonometer. A solution of equal parts of procaine hydrochloride and epinephrine hydrochloride was injected to the quantity of 2 cc., retrobulbarly. Tonometric readings were made one minute, four minutes and then every five minutes after the injection. Curves and tables of the results accompany the article. The authors conclude that (1) in 80 per cent of the eyes there was an immediate lowering of tension; (2) that the most favorable moment for operation is ten minutes after the injection, when the curve of the hypotonia is at its lowest level, and (3) that lowered tension is an additional means of security in total extraction.

L. L. Mayer.

Pharmacology

A Laboratory Study of Some Antiseptics with Reference to Ocular Application. R. Thompson, M. L. Isaacs and D. Khorazo, Am. J. Ophth. **20:** 1087 (Nov.) 1937.

Experiments on the disinfection of living tissue were made with 10 different drugs, and the following summary and conclusions were given:

"The following properties of a number of antiseptics were determined: The disinfectant rate, under conditions simulating as closely as possible those which occur in human tears, of the highest concentrations nonirritating to the conjunctiva; the influence of increased protein concentration on the disinfectant rate; the influence of dilution of the antiseptic on the disinfectant rate; and the toxicity of the antiseptic for leukocytes and for lysozyme.

"The efficacy of the various antiseptics in removing organisms from the conjunctiva is certainly in great part determined by the properties listed above. We are not yet, however, in a position to weigh accurately the importance of the various positive or negative values and to say that one substance is superior to another; to say, for example, that a marked disinfectant action is or is not counterbalanced by an extreme toxicity for leukocytes. It is evident that the circumstances under which the agent is to be used would alter the weight given to the various properties. With repeated application in the case of an infection it is likely that leukocytic injury would be more detrimental than in removing organisms from the membrane with one application previous to operation."

W. S. Reese.

The Pupil

PUPILLOMETRY AND ANISOCORIA. J. A. PEYRET, Arch. de oftal. de Buenos Aires **13:** 16 (Jan.) 1938.

Experiments are here detailed of pharmacodynamic tests as recommended by Sciortino for the determination of the physiologic or pathologic nature of anisocorias and for the testing of the pupillary reaction in normal and abnormal conditions and of the pharmacodynamic reaction of the pupil to drugs which produce contraction or dilatation. The following conclusions are reached: 1. Normally, cocaine hydrochloride (0.25 per cent) produces a mydriasis of from 0.25 to 0.5 mm.; pilocarpine nitrate (1:3,500), a miosis of from 0.5 to 1.75 mm., and physostigmine sulfate (1:6,000), a miosis of from 1.75 to 2.25 mm. 2. Figures outside these limits point to a dysfunction of the dilator (sympathetic) or constrictor (parasympathetic) innervation. 3. In physiologic anisocoria, both eyes react equally and within the normal limits. 4. In pathologic anisocoria, each pupil reacts differently as regards the degree of contraction or dilatation, these being above or below normal. 5. In cases of tabes in which the fundus was normal, Peyret has found a hypoexcitability of the dilator and a hyperexcitability of the constrictor. 6. Even when there is no anisocoria, the tests are useful, allowing the observation of slight variations in the pupillary reactions. 7. The employment of weak solutions is of great importance in carrying out these tests. 8. The examination of the pupils should be made in the darkroom and with the same intensity of light for each eye, and a pupillometer should be employed that registers differences of 0.25 mm. 9. In the observation of the dilating and contracting tests, a period of twenty-four hours should be allowed to elapse between the two tests. 10. The author considers these tests of use in neurologic and clinical examinations in cases in which some disturbance of the sympathetic nervous system is suspected. A report of 65 cases follows.

C. E. FINLAY.

Physiology

SENSITIVITY OF THE PERIPHERAL PORTION OF THE RETINA FOR AVERAGE BRILLIANCE. J. ESCHER-DESRIVIÈRES, Bull. Soc. d'opht. de Paris **50:** 7 (Jan.) 1938.

By means of an instrument devised to compare brilliancy at the macula and periphery, the author has experimented with reactions to various colors of differing intensity of light. The extramacular region is poor in its ability to contrast form and likewise color. However, the author believes that the loss of appreciation by the peripheral elements is one of quantity and depends entirely on the amount of illumination. The fovea is particularly less sensitive to blue than other portions of the retina. He disagrees with the view that nocturnal vision is a function of the rods while day vision is controlled by cones, believing rather that the fovea is the specific organ of clear vision.

L. L. MAYER.

THE FOVEAL MOSAIC. E. P. FORTÍN, Arch. de oftal. de Buenos Aires 13: 51 (Feb.) 1938.

This paper does not lend itself well to abstracting. In it Fortín describes and illustrates his observations on the foveal region, with his peculiar entoptic methods—methods which he has been using for the study of the retina for the last thirty years. The apparatus he uses consists of a Cooper Hewitt mercury vapor lamp at its maximum illuminating intensity, an opaque screen with an aperture of 3 cm., a cubic vessel with parallel walls containing a blue liquid and a disk with a stenopeic opening such as is found in trial cases. The rays from the lamp are concentrated by lens on the 3 cm. aperture of the screen; the stenopeic opening is moved behind the blue filter. At the present time Fortín replaces the vapor lamp with a Philipps mercury lamp, which is easier to obtain and more moderate in price. In any case, it suffices to take a stenopeic disk from the trial case and move it before the eye, which should fix a homogeneous cloud of violet blue through the branches of a tree or an electric lamp with an opaque bulb.

Contrary to what one would expect from optical reasons, mydriasis increases the clearness of the image. Fortín suggests the following possible explanations for this: mechanical action by the nearing of the reticulum under observation to the perceiving screen; physicochemical action, mydriasis producing movement of pigment and alteration in the secretion of retinal pigment, and the possibility of the cones communicating with the granules in the dark.

The fovea is of great importance in relation to visual acuity, and among the visual cones there must be one which functions as the fixation cone, situated at the termination of the axis of retinal coordination.

Fortín describes foveal cones 73 microns in length and 2 microns in diameter; he thinks that their length varies with light and darkness. He describes a peculiar formation in man and in primates in the membrana limitans externa, the fovea or foveal cup, with long diverging regularly disposed fibers 500 microns in length, to which he attributes great importance.

C. E. FINLAY.

Retina and Optic Nerve

A MELANOSOME-DISPERSING SUBSTANCE IN THE BLOOD AND URINE OF PATIENTS WITH RETINITIS PIGMENTOSA (PRELIMINARY COMMUNICATION). E. C. DAX, Brit. J. Ophth. 22: 345 (June) 1938.

A substance was found in the blood and urine of 8 adult mental defectives with retinitis pigmentosa which was capable of dispersing the melanosomes of the frog. It could not be detected in either the cisternal or the lumbar cerebrospinal fluid by the method used.

Urine containing a similar substance has not been obtained from any other patients except certain pregnant women and some persons with abnormal pigmentation of the skin, hyperthyroidism or pituitary disease. Three of the eight patients had signs of pituitary dysfunction in addition to retinitis pigmentosa.

The author's summary follows:

"The blood and urine of eight mental defectives with retinitis pigmentosa contains a substance which will disperse the melanosomes of the frog.

"A similar effect can be produced by the urine when there is pituitary abnormality or when the gland is subjected to physiological stress, but not in other conditions.

"The evidence suggests that the substance in the blood and urine from these cases of retinitis pigmentosa is of pituitary origin."

W. ZENTMAYER.

RECURRING RETROBULBAR NEURITIS: REPORT OF TWO CASES. A. ADAMANTIADIS, Ann. d'ocul. **175:** 432 (June) 1938.

Relapses in cases of retrobulbar neuritis are not rare, particularly in those of neuritis of syphilitic origin. We have wondered whether the primary malady, generally syphilis or tuberculosis, which the patient is suffering from in most instances and which has caused other ocular manifestations is always at the root of these relapses, and it is in connection with this side of the question that 2 cases are reported in detail.

The first was that of a woman of 48 years, with chorioretinitis and atrophy, in whom a first examination twenty-two years previously revealed a positive Wassermann reaction but in whom the present examination of the blood and cerebrospinal fluid gave negative results. During a fourteen year interval, eight relapses of retrobulbar neuritis occurred in the same eye. Antisyphilitic treatment did not seem to affect the course or prevent the relapses. In the interval between attacks the patient had two attacks of iritis, which cleared up quickly.

The second case was that of a man, probably syphilitic, who showed signs of old tuberculosis. He had had seven relapses in one eye during the course of ten years and in addition had had manifestations in the uveal tract preceding the neuritis and between the recurrences.

S. H. MCKEE.

OBLITERATION OF THE RETINAL ARTERIES AND RETINAL ARTERIAL HYPERTENSION. J. BISTIS, Ann. d'ocul. **175:** 447 (June) 1938.

The causes of retinal arterial hypertension are known and have been described. In general hypertension the retinal arterial pressure is elevated, the two tensions as a rule being generally related. In albuminuric retinitis the retinal arterial tension is also much elevated. A similar hypertension of less degree occurs in persons with diabetic retinitis as well as in aged persons with hemorrhagic retinitis. Syphilis is a frequent cause of retinal arterial hypertension. A case is reported in which retinal arterial hypertension occurred with obliteration of the vessels of the retina.

Mme. S., aged 47, had normal urine, a negative Wassermann reaction and a general arterial tension of 21/13. The vision of the right eye was 7/10; the contracted field showed a paracentral scotoma. The superior temporal arteries were transformed into whitish threads. Vision in the left eye was 1/10; the left visual field was retracted similarly to the right, and here, too, there was a paracentral scotoma. The superior temporal arteries appeared as white bands. The disk was atrophic, and there were numerous retinal hemorrhages.

The author concludes that with the obliteration of the branches of the central artery of the retina there was an increase in retinal tension, probably influenced some by the general hypertension.
S. H. McKEE.

Trachoma

THE WORLD-WIDE DISTRIBUTION OF TRACHOMA EXCLUDING THE DOMINIONS, COLONIES AND MANDATED TERRITORIES OF GREAT BRITAIN. A. F. MACCALLAN, Brit. J. Ophth. **22:** 513 (Sept.) 1938.

The areas excluded in the title were considered in the November 1934 issue of the *British Journal of Ophthalmology* (page 625). The present dissertation is directed to the distribution of trachoma in other parts of the world. Information as to the incidence of trachoma in Afghanistan, Albania, Abyssinia, Colombia, Costa Rica, Haiti, Honduras, Liberia, Nicaragua, Panama, Paraguay, Salvador, San Domingo and Siam was not obtained.

The author has succeeded in placing before the ophthalmologist in readable form an approximate estimate of the high incidence of trachoma which exists throughout the world.

It is not possible in an abstract to give the figures for the different countries, but attention is called to this excellent résumé and its value for reference.
W ZENTMAYER.

Uvea

IRIDOCYCLITIS AND FOCAL INFECTION. H. LAGRANGE and J. GOULESQUE, Ann. d'ocul. **175:** 493 (July) 1938.

In 1927, and again in 1934, reports of cases of optic neuritis in which cure followed treatment of a maxillary sinusitis were reported by one of the authors. There was also reported a case of acute iridocyclitis in which the condition resisted all orthodox treatment but was at last cured after the removal of a dental cyst. The authors had an opportunity to study 3 new cases of iridocyclitis associated with sinus infection. These are reported in detail.

In the first case the patient had a positive Wassermann reaction, which brought up the question of syphilitic iritis. A sinusitis had developed which did not present any subjective or objective signs apart from a low grade rhinitis. Similarly, in the second case the Wassermann reaction was also positive and there were no evident roentgenographic signs. In the third case the etiology was somewhat more obscure after syphilis, tuberculosis and rheumatism were ruled out. In all the cases the most notable feature was the rapidity with which pain ceased after treatment of the sinusitus. This is important and agrees with the course in the cases reported in the earlier articles.

It is without doubt necessary to modify the ideas with regard to iritis, which for twenty-five years has been considered as commonly caused by syphilis. A typical syphilitic iritis is a manifestation of secondary syphilis, and with the present methods of treatment secondary syphilis is now seen much less frequently. An extensive bibliography accompanies the article.
S. H. McKEE.

Therapeutics

A Hydrostatic Approach to the Posterior Chamber for Diagnostic and Therapeutic Purposes. A. Motegi, Brit. J. Ophth. 22: 543 (Sept.) 1938.

Intraocular irrigation is of use in patients with poor vision in whom the anterior segment of the eye is nearly normal but the fundus is invisible; in patients with poor vision following diseases of the anterior segment, peculiar to the tropics, in whom the fundus is invisible (in both these types a view of the fundus would be of great value), and in persons with panophthalmitis for the extraction of nonmagnetic foreign bodies from the eyeball.

The author describes the necessary incision of the eyeball and the apparatus required. The solution used is 0.9 per cent salt solution or Ringer's solution.

Reports of several cases are included, and the article is illustrated.

W. Zentmayer.

Society Transactions

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HARRY S. GRADLE, M.D., *President*

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WILLIAM P. WHERRY, M.D., *Secretary*

OPERATIVE TREATMENT OF RADIATION CATARACT. DR. ALGERNON B. REESE, New York.

This article is published in full, with discussion, in this issue of the ARCHIVES, page 476.

PECULIARITIES OF WELL KNOWN OCULAR DISEASES IN THE NETHERLAND EAST INDIES. PROF. A. W. MULOCK HOUWER, Batavia, Netherland East Indies.

This article was published in full in the February issue of the ARCHIVES, page 235.

OSSIFICATION OF THE CHOROID. DR. BERNARD SAMUELS, New York.

The material that formed the basis of this study consisted of microscopic preparations from 81 ossified globes, enucleated mostly because of atrophy, phthisis or secondary glaucoma. As only a collection of ossified globes was studied, it is not possible to give exact data as to what percentage of all atrophic globes enucleated contained bone. In many enucleated globes the atrophy is of recent origin, enucleation having been done early for fear of sympathetic ophthalmia. One does not expect to find ossification in globes with recent atrophy. A study of globes with long-standing atrophy in which injury or disease took place a number of years before enucleation would probably show that about one third of them contained bone. However, globes that have been atrophic a great length of time are seldom enucleated, especially when atrophy has followed metastatic ophthalmitis or spontaneous iridocyclitis. In the latter cases the fear of sympathetic ophthalmia is not great. Such eyes seldom become inflamed. That ossification is not rare in atrophic globes is suggested by the fact that the present survey deals with such a great number of cases.

A history of perforation of the globe was obtained for 37 of 78 globes. Corneal lesions had affected the interior of 16 globes. Endogenous diseases, such as spontaneous uveitis and metastatic ophthalmitis, were found in 25 globes. The globes showing retinal detachment

were especially interesting because apparently toxins formed locally in the subretinal fluid were a factor in the production of bone.

In a general review of the material it was possible to differentiate three types of bone formation according to shape: first, plates of solid bone in various locations; second, thick shells of bone, occasionally measuring as much as 7 mm. in thickness; and, third, thin spicules of bone, frequently not calcified and lying most often within the choroid or beneath the pigment epithelium. Bone marrow was associated with the second type of extensive shell-like formation at the papilla. It usually lay enclosed in an elaborate reticulum of long slender bone trabeculae lying parallel to the surface and connected with one another by still more delicate bridges of bone. The marrow was made up of a meshwork of granulation tissue, in which scattered lymphocytes or lymphocytic nodules were found, together with many endothelial tubes. In the interstices of the meshwork a large amount of fat cells was found, forming typical yellow bone marrow. Fat in the globe is associated with bone trabeculae and almost never occurs independently. Marrow was never encountered in the flat and spicule types of bone, which reminded one of the cranial bones. The presence and the amount of marrow were directly related to the amount of bone.

Certain places showed a definite predilection for bone. Sometimes there was just one isolated piece of bone. In other instances the bone was distributed on more than one place, this being termed "combined localization." The most common localization was found to be on the surface of the choroid, in the posterior quadrant of the globe, as is mentioned in the literature. From this location it more than once extended forward over the equator to unite with a ring of bone that grew backward from the ora serrata. In the coalescence of the two zones a complete cup of bone was formed. A small hole posteriorly was invariably left for the passage of the atrophic nerve fibers. The ring of bone at the ora serrata, which was generally not so complete as the one surrounding the nerve head, often possessed irregular jagged margins from extensions inward along cyclitic membranes. As is well known, the growth of bone may be so extensive as to occupy almost the entire cavity of a shrunken globe.

Bone formation within the lens was encountered seven times, and a few times bone was found in the circumlental space. In every instance in which bone was found in the lens, on the ciliary body or at the ora serrata it was combined with bone elsewhere. Ossification of the lens may be best explained in the mode accepted in the literature, that is, by the extension of connective tissue into the capsule. This may happen when no injury has destroyed the lens capsule. In a case of metastatic ophthalmitis in the third month of life, toxins dissolved the posterior capsule of the lens, permitting connective tissue to enter from a cyclitic membrane. That really connective tissue was the basis of the ossification and not epithelial fibers derived from the capsular epithelium was proved by the presence of blood vessels in the midst of bone lamellae that substituted lens matter.

Bone on the surface of the ciliary body as a rule did not give the impression of having been a transformed connective tissue membrane. The bone lamellae always lay on the surface of the epithelium itself and more than once even separated a membrane from the ciliary body. As

a matter of fact, direct evidence of the transformation of a dense connective membrane into bone was lacking everywhere as a general rule. On the contrary, a narrow stratum of loose fibroblasts often separated the bone from a denser connective tissue membrane.

The retina showed bone formation in 5 cases. In 2 of these extensive ossification of the choroid was combined with small pieces of bone in the retina near the ora serrata. The ora serrata is a favorite site for different kinds of reactive proliferation, as, for instance, migration of the pigment epithelium into degenerated and detached retinas. It is, therefore, not astonishing that bone was found here at the retinal attachment to the ora serrata. Bone within the retina differed from that on the choroid, presenting more jagged outlines and being more irregularly calcified. The bone corpuscles were much scantier, rendering the differentiation from pieces of chalk difficult.

A comparison of the distribution of the bone in various parts of the globe brought out that there are two favorite sites of predilection: one in the posterior quadrant, especially around the papilla, and the other at the ora serrata. Predilection for the region of the papilla held good as well for small pieces of bone in the choroid as for large shells of bone on its surface. It was at the papilla that large shells of bone attained their greatest thickness. Sometimes, pulled forward by traction, the detached retina dragged the papilla and the adjacent choroid with it, and in the drawn out atrophic part of the choroid bone was sometimes found. Bone in the immediate neighborhood of the papilla lay not only on the surface of the choroid but frequently in the choroid itself, in uninterrupted contact with the optic nerve. At the second place of predilection, the ora serrata, bone was found only when the attachment of the retina was confined to the immediate neighborhood of the ora serrata, by which is meant that the retina remained in direct contact with the choroid at this point. This condition could not have been fulfilled had the detached retina, by taking with it the ciliary epithelium, extended forward along the ciliary body. In the latter event only a delicate film of epithelial cells would have connected the detached retina to the uveal tract, and there would have been no traction.

Traction is a most important stimulus to the production of bone in various parts of the globe. The subretinal fluid, although it is assigned as a cause for the production of callus, does not seem to play the same role in the production of bone at the ora serrata. There was never any certain indication that the fibroblasts that were supposed to lay down bone were produced from the pigment epithelium.

According to this study, it may be affirmed that the danger of sympathetic ophthalmia from globes ossified after perforation is practically nonexistent. It is instructive to recall that in an examination of over 100 globes with sympathetic ophthalmia there was not a single instance of ossification. It must be extremely rare, if it ever does occur, that ossification and sympathetic ophthalmia happen in the same globe.

Turning to sarcoma of the choroid, I venture to affirm that sarcoma is as little likely to develop in atrophic and shrunken ossified globes as is sympathetic ophthalmia. This statement is based on the fact that in an examination of 84 globes with necrotic sarcoma there was no one instance of ossification; it mattered not whether the globe had become atrophic before sarcoma developed or had become so afterward.

DISCUSSION

DR. BRITTAIN FORD PAYNE, New York: It has been my good fortune to study most of the specimens described by Dr. Samuels. From his masterful work on ossification of the choroid, at least two observations may be made:

1. Bone formation in the choroid has little or no bearing on sympathetic inflammation of the other eye. This seems startling in that Fuchs and others advised immediate removal if any sign of irritation or tenderness is present.

2. Bone formation is absent in cases of necrotic intraocular sarcoma. This led to an investigation of other intraocular neoplasms in the collection of globes at the New York Eye and Ear Infirmary. It was observed that bone was absent in cases of intraocular sarcoma and carcinoma, whether the growths were necrotic or not. Globes with necrotic glioma of the retina showed deposits of calcium.

An examination of specimens affected with ocular tuberculosis and syphilis failed to show bone formation. Hyalinization and calcification were not uncommon.

The architecture and anatomic features have been excellently described. The development and location of the lesions have been explained, but the actual cause of bone formation remains as much a mystery as it was a century ago. Physical, chemical and biologic factors must be involved. It is possible that stress and pull on membranes are responsible, but it must be remembered that low carbon dioxide tension may be a factor. Saponification of fatty acids as shown by Klotz may offer some explanation. However, all of these agents in addition to others more complex probably are the answer.

Repeated investigations have shown that hyalinization, calcification and bone formation occur in dead or devitalized tissues. According to MacCallum, the kidneys, lungs, spleen, liver, heart, blood vessels and subcutaneous tissues may be affected. No definite explanation has been found for these changes in any of the organs named. The ophthalmic literature fails to give a definite clue.

Fortunately, such heteroplasia is self limited in the eye. If bone should extend through the choroid and sclera and involve the adnexa, the ophthalmologist would be greatly astonished and the patient would be uncomfortable.

The question finally arises, should a blind atrophic eye be removed? It should be enucleated if there is pain or irritation or if the other eye is affected.

DR. MARY KNIGHT ASBURY, Cincinnati: In reviewing several hundred eyes examined microscopically within the last three years, I was surprised to find bone in more than 10 per cent of them. The series was much smaller than Dr. Samuel's, but bore out his findings in detail—the predominance in males (19 males and 4 females in my series), the wide age distribution (at the time of enucleation the youngest patient in my series was 3 years old and the oldest 65), the types of bone found and the location—and in addition furnished interesting exceptions, of which I shall speak later.

I agree with Dr. Samuels that neither calcium nor visible membranes are necessary preambles to the formation of bone in the eye. Bone has been found in scar tissue, in cyclitic membranes and in eyes which also contain calcium deposits, but it is also seen in the choroid beneath the pigment epithelium, with little evidence of scar tissue formation around it. However, all the eyes in which bone is found have been severely injured or inflamed, and at least a minimal fibroblastic response is present. When the versatility of mesodermal cells is remembered, the production of bone ceases to be so mysterious. The young cell, which usually produces fibrous tissue, may find the conditions favorable for bone formation and develop that potentiality.

I shall now consider the exceptions afforded by my specimens.

Dr. Samuels described "oral bone," an osteoid deposit in the region of the ora serrata, and stated that such a deposit was never found without the presence of bone elsewhere in the globe. In my series a small "oral bone" was found in the eye of a child aged 4 years who had had an abscess of the vitreous complicating meningitis eight months before enucleation. (This is the shortest duration of a lesion in which bone was found in the series.) No evidence of ossification was found elsewhere in the eye. In another eye the only osteoid tissue found was in the anterior part of the globe in the midline in a mass of scar tissue between the iris and the detached retina. In this instance the tissue immediately surrounding the bone was loose and vascular, as described by Dr. Samuels.

Both malignant melanoma and bone were found in one specimen. The eye had been blind for twelve years because of retinal detachment of unknown etiology.

I also doubt that ossification has any real bearing on the cause of sympathetic ophthalmia, but my series was less conclusive on that point than that of Dr. Samuels. An eye had been shrunken for fifteen or twenty years, no further history being obtained. The retina had long been detached, the lens was calcified, and there were coarse granular deposits of calcium on the surface of the ciliary body. A plaque of bone was found in the choroid at the margin of the disk and in the adjacent retina. Much of the choroid had been destroyed, but there was active inflammatory infiltration of the remainder, which was suggestive of sympathetic uveitis. Dr. Terry saw these sections about a year ago and concurred in the diagnosis. Recently a small shrunken globe was examined which had been blind for forty years. A thick shell of spongioid bone filled much of the vitreous cavity. Contrary to rule, the bone was thicker anteriorly, and the shell was incomplete around the disk. The calcareous lens was embedded in fibrous scar tissue. In the center of the dense band occupying the anterior chamber there was a large plaque of osteoid tissue. Dr. Samuels stated that ossification is never seen in the anterior chamber. The remains of the uveal tract was heavily infiltrated with inflammatory exudate resembling that found in sympathetic uveitis.

Ocular Papilloma. Dr. R. E. Windham, San Angelo, Texas.

This report pertains only to cases of ocular papilloma clinically diagnosed and confirmed by pathologic examination. The series includes

8 cases in which cure was obtained and 1 in which enucleation was done. Papilloma of the conjunctiva is a comparatively rare condition and frequently misdiagnosed or confused with other pathologic conditions. The majority of cases reported have involved the caruncle and the palpebral conjunctiva, but this report is limited to cases in which papilloma appeared on the ocular conjunctiva and cornea. The etiology of papilloma is an unsettled question. Some ascribe the condition to trauma. I am convinced that in the adult papillomas are due to trauma from wind containing dust and grit. In all cases the papilloma appeared on developed or potential pterygium, which condition also is caused entirely by exposure.

Papillomas usually occur in one or the other of two forms, a diffuse papillomatous form or a pedunculated form. The latter has to be distinguished from granuloma, tubercle, vernal conjunctivitis, carcinoma, lymphangioma, xerosis, epithelial plaques and pterygium. Careful resection of the papilloma and cautery of the denuded base will result in a fair number of cures. However, the Shahan thermophore should be the method of choice, since papillomatous tissue in the eye melts away as if by magic at from 150 to 160 F.

DISCUSSION

Dr. Robert J. Masters, Indianapolis: I have not encountered a papilloma of the lobulated type, although it seems that this variety should offer less difficulty in its clinical diagnosis than the sessile type. Even in the microscopic section a flat papilloma of the bulbar conjunctiva may be hard to distinguish from an epithelioma of low grade malignancy. Clinically, several kinds of epibulbar tumors may look much alike and resemble a papilloma of the sessile type. To illustrate this point, I have chosen the following case histories for brief presentation.

In the first case there was a flat, smooth-surfaced tumor of the bulbar conjunctiva of the left eye, extending from the semilunar fold outward and upward and downward, so that the entire nasal half of the conjunctival surface was affected. The tumor disappeared after two treatments with the Shahan thermophore two weeks apart, a general anesthesia being administered during treatment.

The second case was that of a man aged 42, with a flat, oval tumor, measuring 6.5 by 5 mm., on the left eyeball. This tumor followed an injury to the eye. The Shahan thermophore, at 145 F., was applied for one minute. Two weeks later the remaining mass, 4 by 5 mm., was excised. The pathologist reported a small bit of squamous epithelium and granulation tissue underlying loosely organized connective tissue, with all of the underlying tissue presenting the features of a proliferative inflammatory process. This tumor was probably a granuloma, although its surface seemed to be fully and smoothly covered by epithelium.

The third case was that of a man 68 years of age, in whom a small elevation developed on the temporal limbus of the eye following a subconjunctival injection of a solution of mercuric cyanide (1:6,000). The thermophore, at 158 F., was applied twice for one minute, with satisfactory result.

The fourth case was that of a girl 19 years of age, who four months previous to her examination had noticed a fleshy growth on the nasal aspect of the left eyeball. Later, there appeared a second growth on this eyeball and two similar lesions on the right eyeball. The clinical diagnosis was papilloma. Some of the lesions were excised for pathologic examination; others were treated directly with the thermophore. The pathologic examination led to a diagnosis of epithelioma of low grade malignancy. The pathologist's description, however, strongly suggested papilloma. There was a vascular core surrounded by a small amount of fibrous tissue containing many lymphocytes with a covering of several layers of squamous epithelial cells, which infiltrated the underlying structures at some points. The cause of the condition was subject to conjecture, but the patient had used mascara on her eyelashes in profuse amounts for many months.

DR. W. R. BUFFINGTON, New Orleans: According to statistical data, malignant, or recurring, ocular papilloma is relatively rare. To determine the exact frequency, careful clinical diagnoses should be made as well as precise histologic study. The case reports so well given by Dr. Windham show that this type of papilloma is far more frequent in southwest Texas than it is in Louisiana. In that section the climate is dry and the wind is heavily laden with dust and sand. In Louisiana moisture free from dust fills the surrounding atmosphere. It would seem that conjunctival irritation from exposure to such substances is a most important etiologic factor.

My statistical reports show that recurring papilloma is extremely rare in Louisiana. Among 14,686 patients with ocular disease admitted to the Charity Hospital from 1922 to 1937, there were only 11 authentic cases of ocular papilloma. These facts would suggest that climatic conditions may account for the relative infrequency of conjunctival papilloma.

Papilloma has certain characteristics which distinguish it from other ocular conjunctival tumors. Usually papillomas are small, flat tumors with uneven or velvety surfaces, composed of branching papillae of connective tissue surrounded by a thick layer of stratified epithelial cells. Conjunctival papilloma invariably recurs unless properly treated. True papilloma never gives rise to metastasis. It may undergo malignant or carcinomatous degeneration. Treatment consists in excision of the epithelioma, after which the denuded area must be treated by some cauterizing agent. This agent may be the electric cautery, the thermophore or a chemical such as glacial trichloroacetic acid. In my hands, the last has been the most efficient.

DR. EDWARD STIEREN, Pittsburgh: A cauliflower growth developed in the right eye of a man 45 years of age. It took on a mushroom shape and was 12 mm. in length, 8 mm. in width and about 10 mm. high. It reached to the external canthus, overlapped the cornea to its center and projected forward between the lids, which could not be closed over it. The surrounding conjunctiva was inflamed and thickened, and there was considerable ropy discharge. The color and general appearance of the mass were those of a pale red raspberry. There was no involvement of the preauricular glands or any of the lymphatic glands. According

to the patient's statement, the lesion was first noticed about a year previously as a localized inflammation with a slight elevation.

The growth was excised and the defect covered with a sliding conjunctival flap. At only one point, over the insertion of the external rectus muscle, was it adherent to the globe. Healing was prompt and uneventful. The specimen was pronounced to be an epithelioma of the papillomatous type or a papilloma underlying epitheliomatous changes. The patient was given roentgen irradiation at intervals over a period of six weeks, and when he was seen a year later the entire conjunctiva was smooth and glistening, with no semblance of a recurrence.

Dr. Norman W. Price, Niagara Falls: R. J. came to my office on Aug. 12, 1937, with a growth on the 9 o'clock line at the limbus of his left eye. It was a flat growth, extending a short distance over the cornea. I removed it with a cataract knife and found it to be only slightly raised and to contain no pedicle. It was sent to the laboratory at the Memorial Hospital, Niagara Falls, and also to the Gratwick Cancer Laboratory, Buffalo. According to the report, the growth was a papilloma. The patient returned in two weeks with another growth as large as the previous one. I took him to the Gratwick Cancer Laboratory. An attempt was made to remove the growth with an electric cautery on different occasions, but it returned soon after each treatment. On November 10 the growth was much in evidence. On March 7, 1938, at the hospital, I removed it thoroughly with a knife and cauterized the base thoroughly with tricholoroacetic acid, covering the area with a conjunctival flap. Another biopsy at this time showed a papilloma. A month later the growth was as large as ever, and as Gratwick refused to use radium, I did a thorough job at cauterizing, burning the tissues all I thought the eye could stand and again covering with conjunctiva the best I could. Since then there has been no recurrence. Vision corrected is 20/30.

Dr. Lawrence T. Post, St. Louis: I agree with Dr. Windham as to the value of the thermophore in the treatment of tumors of the type described. My associates and I have been using the thermophore in our office in these cases, and also in the clinic at Washington University for the past twenty years. We have had excellent success and few recurrences. I believe that the reason one has fewer recurrences in treating with the thermophore is because heat from the thermophore penetrates farther than other types of cauterization, though as Dr. Windham said, penetration probably is limited to about 1.5 mm.

I take exception to his statement about malignant growths. We have found that they were equally successfully treated by the thermophore. We find that the success depends on the condition of the tissue with regard to its relationship to scar tissue. The softer and newer the growth, the more successful is the thermophore application.

Practical Use of Homatropine-Benzedrine Cycloplegia. Dr. Lyle S. Powell, Lawrence, Kan.

Homatropine and benzedrine used in combination will produce in sixty minutes complete practical cycloplegia in a high percentage of

patients between the ages of 16 and 31. Beginning recovery is evident in four hours. A 1 per cent or 0.5 per cent solution of physostigmine salicylate, buffered to be isotonic with tears, will overcome cycloplegia produced by homatropine and benzedrine promptly and restore the power of accommodation within one-half hour.

The technic used by me is as follows: Two drops of a 2 per cent solution of homatropine hydrobromide are instilled into the conjunctival sac five minutes apart, followed by 2 drops of a 1 per cent ophthalmic solution of benzedrine sulfate, five minutes apart. The results obtained in parallel groups of persons with the orthodox homatropine method and with the homatropine-benzedrine method have been tabulated and compared. Only a small number of the patients given homatropine showed a complete return of accommodation at the end of eighteen hours. The groups given homatropine and benzedrine had complete return of accommodation at the end of eight hours, with a number of persons showing complete return of accommodation at the end of four hours. The pupils were slightly larger and the corneas definitely clearer in the cycloplegic stage in the groups given homatropine and benzedrine than in the groups given homatropine alone.

DISCUSSION

Dr. C. W. Rutherford, Indianapolis: Studies have been made during the past year on the effects of benzedrine and paredrine when added to homatropine for cycloplegia and on the significance of variations in the location of the far point (*Tr. Am. Acad. Ophth.* 42: 188-189, 1937). Both eyes of 174 private patients, including a few young persons with myopia, were examined.

The patients studied were divided into 6 groups for comparison. The type and amount of cycloplegia were different for each group, the drug used being homatropine hydrobromide, to which was added either benzedrine sulfate or paredrine hydrobromide in various amounts. It became evident that human variables were responsible for variations in diopters and that great care was necessary in conducting these tests. Close attention to the behavior of patients whose residual accommodation was being tested revealed that some persons spoke much more quickly than others, while some persons read square letters earlier than round ones and others read round letters earlier than square ones. This series of studies revealed a variation of 0.25 diopter from 33.3 cm., which is an expression of variability in human behavior or aptitude, and it cannot be eliminated. There can be no uniform precise location of the far point. Benzedrine and paredrine did not augment the degree of cycloplegia obtained with homatropine alone; both increased mydriasis, which was of no apparent advantage; both shortened the duration of accommodative disability, which was a welcome convenience to the patients.

Dr. S. Judd Beach, Portland, Me.: The experience of my associate and me with benzedrine and paredrine in cycloplegia leads us to believe that it does not make a great deal of difference whether one employs the conventional method of repeating the instillation with the idea of getting cumulative action or the single instillation method, as Dr. Powell has described, reenforced with adrenergic drugs. Also,

we are inclined to think that it makes relatively little difference whether one uses a 1 per cent solution of benzedrine sulfate or a 1 per cent solution of paredrine hydrobromide, or, as has been recently recommended, paredrine hydrobromide in a 3 per cent solution. The result seems to be about the same if the solutions are combined. At first we were firmly convinced that one drug was distinctly better than another, but after we compared the results in the same eyes at different times, using different drugs, we found that the variation which at one time indicated that one drug was better, the next time might show that the other drug was quite superior; and paradoxically enough, sometimes we found for the same patient that a 1 per cent solution of paredrine hydrobromide, for instance, had apparently been more effective in the same combination with a cycloplegic than a 3 per cent solution, and this has been more or less confirmed, I think, by Tassman's experience with paredrine, which he recently published.

I like Dr. Powell's phrase "practical cycloplegia," because cycloplegia which one ordinarily gets and which is quite satisfactory for the purposes of refraction is, as he said, "very remote from complete cycloplegia."

The point where our experience diverges from that just reported by Dr. Powell is in the use of homatropine alone and atropine alone for children. I place no reliance whatever on homatropine alone used in the conventional way for children of school age or, in fact, for any patients under 16 years of age; and, conversely, I feel that one of the greatest benefits and comforts to patients that I have had has been in the combining of the adrenergic drugs with atropine for patients under 16 years of age.

We can certainly confirm Dr. Powell's experience with regard to the use of physostigmine salicylate. The use of this drug after the ordinary conventional use of homatropine is followed by apparent prompt recovery from the cycloplegia, but the effect of the drug soon wears off and the effect of the homatropine returns. In cases, however, in which the action of the physostigmine salicylate is maintained approximately as long as the action of the cycloplegia, the quick recovery is sustained.

Dr. William Crisp, Denver: The combination which I personally have been using since I learned of the experiences of Dr. Beach and McAdams has been as follows: I use, first, a 2 per cent solution of cocaine hydrochloride; four minutes later I use a 1 per cent solution of benzedrine sulfate or, more recently, because the manufacturer seems to think that paredrine is a little better than benzedrine and is sending out the sample preferably of paredrine, a 1 per cent solution of paredrine hydrobromide. Four minutes later I instill 1 drop of a 5 per cent solution of homatropine hydrobromide. I am disposed to think that my experience has borne out Dr. Beach's statement that one gets better results with 1 drop of the stronger solution of homatropine hydrobromide than with the 2 drops of the weaker solution. I used to employ 2 drops of the 2.5 per cent solution.

I have found this combination to yield satisfactory cycloplegia after one hour. If the drops are all placed on the sclera above the cornea, with the upper lid held out of the way, I do not believe that the objection

as to the preliminary use of paredrine shrinking the lacrimal passages has much validity.

DR. I. S. TASSMAN, Philadelphia: I began the use of benzedrine sulfate in aqueous solution in addition to homatropine hydrobromide following the work of Drs. Beach and McAdams, and in the same manner I completed cycloplegia in a number of cases. The results that I obtained corresponded closely with the results reported by Dr. Beach.

I began by using benzedrine sulfate in the same strength solution that Dr. Beach reported, the 5 per cent solution, and then experimented in various ways, using weaker solutions, including 2 drops of the 2 per cent solution in the way that was described by Dr. Powell.

About a year and a half ago the effect of paredrine came to my notice, after an investigation by two men at the University of Pennsylvania. I then substituted an aqueous solution of paredrine hydrobromide for the solution of benzedrine sulfate, also trying solutions of various strengths, beginning with 2 drops of a 2 per cent solution, 1 drop of a 4 per cent solution and again 1 drop of a 5 per cent solution. In regard to both the benzedrine and the paredrine, I cannot say that the results I obtained were as satisfactory with 2 drops of a 2 per cent solution as those obtained with 1 drop of a 4 per cent solution or of a 5 per cent solution. I did feel that the results with 1 drop of a 4 per cent solution were practically the same as those I obtained with 1 drop of a 5 per cent solution.

As to children of school age, or below the age of, say, 15 or 16 years, I found that it is perfectly satisfactory to use 1 drop of a 1 per cent solution of atropine sulfate, followed in three or four minutes by 1 or 2 drops of a 1 per cent aqueous solution of paredrine hydrobromide. The results are practically the same as those that are obtained after the repeated instillation of atropine sulfate. As a matter of fact, I believe that one really uses the numerous or repeated instillations in the ordinary way first of all because they are made at home, probably by a parent who does not know any too much about the proper method of instillation, even though it has been explained, and are given principally to insure at least a couple of drops being instilled properly.

MECHANISM OF OPTOKINETIC NYSTAGMUS. DR. NORMAN P. SCALA, Washington, D. C., and DR. ERNEST A. SPIEGEL, Philadelphia.

The mechanism of the subcortical type of optokinetic nystagmus (so-called passive nystagmus) was studied in cats and dogs. The production of this nystagmus depends on the intactness of the superior colliculi, one-sided lesions of this structure impairing the nystagmus to the opposite side, while after bilateral lesions, at the most, rudimentary reactions can be elicited by the optokinetic impulses. After lesions of the vestibular nuclei, various interference phenomena between the "spontaneous" central and the optokinetic nystagmus were observed. The optokinetic impulses may increase the frequency of a spontaneous central nystagmus if it is synergistic in direction; they may diminish its frequency or even reverse its direction if it is antagonistic. Sometimes the jerks of the optokinetic nystagmus may be observed in the intervals between the periodically beating spontaneous nystagmus. Large

lesions of the vestibular nuclei may prevent the appearance of horizontal passive nystagmus in one or both directions, even if the reticulate substance is spared. These experiments suggest that the vestibular nuclei play an important part in the mechanism of the passive nystagmus.

DISCUSSION

Dr. A. Bielschowsky, Hanover, N. H.: In the discussion of their excellent experimental work, Drs. Scala and Spiegel refer to the contradictory statements in the literature concerning the question whether optokinetic nystagmus can be produced only if the subject's attention is concentrated on moving objects, so that optokinetic nystagmus would have to be considered a cortical reaction to the retinal stimuli, or whether it may be regarded as a subcortical reflex, a belief based on observations of optokinetic nystagmus in the newborn, in idiots and in unconscious patients and, furthermore, on experiments showing that passive optokinetic nystagmus may be elicited after extirpation of the cerebral hemispheres in rabbits, dogs and monkeys. Optic fibers conveying retinal stimulation from the primary optic centers (external geniculate body) to the corpora quadrigemina and the oculomotor nuclei have been supposed to represent the pathway for reflex movements of the eyes elicited by retinal stimuli. Whether or not this pathway is made use of not only in animals but in man has been disputed for a long time. Since light perception in man is a function confined to the cortex, it is hardly conceivable that a movement of the eyes may be elicited by a retinal stimulus as a subcortical reflex, namely, without the participation of the occipital cortex in the process. The supposed existence of such a reflex mechanism would involve the assumption that persons with cortical amaurosis could be induced to move their eyes at a certain angle as well as in a certain direction by having light in a dark room thrown onto a certain spot of their retinas. The movement brought about by the reflex mechanism under discussion would bring the peripheral image of the light to the fovea without the person perceiving the light. Not only is this inconceivable, but it never does occur in reality. In 1911 I had under my observation for several weeks and until she died, a patient with complete cortical amaurosis. Autopsy showed a circumscribed destruction of both occipital poles due to the obstruction of the two posterior cerebral arteries. During the observation her mental condition was absolutely normal. Despite the complete blindness, the pupillary reaction to light was perfectly normal, showing the integrity of the reflex pathways connecting the external geniculate body with the oculomotor nuclei. Although the patient was able to execute to the normal extent both the voluntary and the commanded ocular movements in any direction and moreover to direct her visual lines to her own hand or to an object she was touching, no ocular movement from even the strongest retinal stimuli resulted, although she was warned to watch for a light flashing somewhere before her eyes. Such a behavior in a case of cortical blindness, in which postmortem examination confirmed the clinical diagnosis based on the integrity of the pupillary reaction to light and the absence of atrophy of the optic nerve, is an irrefutable argument against the assumption that in man the retinal stimuli conveyed on subcortical pathways, that is, without passing through the cortex to the oculomotor nuclei, may

give rise to true reflex movements of the eyes. It is, of course, different in animals in which visual sensations originate not only in the hemispheres but in the optic lobes, so that ocular movements may be elicited by retinal stimuli after the extirpation of the hemispheres.

DR. W. B. LANCASTER, Boston: I believe that nystagmus deserves more attention than it receives. I look on it as a defect of fixation. This is well illustrated by nystagmus in children with a defective macula, who are therefore unable to fix perfectly. Hence the study of nystagmus will throw light on the physiology of fixation, an important subject.

One should not think of nystagmus as a simple condition due to only one mechanism. Is it not probable that fixation involves many factors acting through many pathways between different parts of the central nervous system, what are often called centers? All these areas and many others are embraced in what I like to call the neuromuscular mechanism for ocular movements. In simplest terms, fixation occurs in this fashion: An image of some object falls on the periphery of the retina. The image attracts one's attention, and a reflex mechanism provides a movement, so that the image falls on the fovea. If the movement, as is usual, is not exact but overshoots the mark, the image falls near the fovea, but beyond it and therefore still peripheral. By peripheral I mean any part of the retina outside of the macula. This requires a reverse movement, and if this again overshoots the mark, going beyond the fovea on the other side, another correcting movement is required. When this is kept up the result is nystagmus.

The following simple conception is my idea of the mechanism of optokinetic nystagmus. When a person observes a series of moving objects passing by, as from a car window, the eye fixes on one object, follows it along and then is arrested by another, to which it jumps back and follows that along. This is repeated indefinitely, as, for instance, in the setup that was described, consisting of a large paper cylinder in which the subject's head is placed and the stripes are observed moving steadily in front of the eyes.

Perhaps the connection of the vestibular apparatus accounts for this regularity, this comparatively *uniform rhythm* with which one moves the eyes back and forth. The vestibular apparatus is an old, one might say, primitive, part of the nervous system. As new reflexes and new activities develop, this mechanism is utilized. Pathways from the central areas, that is, from the frontal area for voluntary movements and from the occipital area for the visual motor reflexes, go through or are connected intimately with the vestibular area, and the performance is affected by this mode of action. In order for the impulses from the occipital visual motor cortical area, for instance, to reach the centers of the third, fourth and sixth nerves, the impulses follow pathways which these authors have studied. They have shown that the vestibular nuclei receive impulses not only from the labyrinth, as originally in the lower animals, but from higher parts of the central nervous system. Thus the connections of the vestibular nuclei with the oculomotor nuclei represent the common forms: (*a*) labyrinthine ocular reflexes, (*b*) voluntary ocular reflexes, (*c*) optokinetic cortical nystagmus and (*d*) optokinetic subcortical nystagmus, at least as far as horizontal movement and horizontal plane are concerned.

SNAKE VENOM IN OPHTHALMOLOGY. DR. M. E. ALVARO, São Paulo, Brazil.

Among the 2,300 more or less known specimens of serpents, approximately 390 are poisonous in a literal sense of the word; but besides these notoriously poisonous snakes, there are others also possessing a veneniferous system, which are not included in the same group because the poison fangs are located in such a position that only by the merest accident can they injure men or animals.

Poisonous snakes belong to three distinct groups:

(*a*) The Opisthoglyphae, characterized by enlarged posterior maxillary teeth, having a longitudinal groove beginning near the excretive canal of the poison gland. These snakes are not generally classified as poisonous (Serpentes suspecti).

(*b*) The Proteroglyphae, or Elapidae, characterized by the anterior maxillary teeth, differentiated and having a partly closed canal communicating with the poison gland.

(*c*) The Solenoglyphae, with movable fangs, articulated and having a completely closed canal, communicating also with the poison gland.

The use of these venoms in the treatment of various diseases is based on the study of their respective physiologic properties, but in some conditions, as for example, epilepsy and algias, the results of their accidental use led the investigators to make further research. Certain venoms increase the coagulability of the blood, and the use of others in the treatment of epilepsy and cancer came from empiric observations of improvement in persons accidentally bitten.

The properties of various venoms have been studied in the recent decades, and they are used according to their physiologic action for a corresponding pharmacodynamic effect. The venoms in which the neurotoxic element predominates are naturally indicated for the treatment of algias because of their soothing effect on sensitiveness. Likewise, the venoms in which the coagulating principle predominates are logically the most appropriate for the therapeutics of hemorrhagic conditions, and the proteolytic factors would naturally be the ones of interest in the use of venom in the treatment of malignant tumors.

To reduce the toxicity of the venoms, various methods have been employed, which, while diminishing their toxic and irritant force, still retain the useful properties. Among them is the weakening of the venom by formaldehyde, as Raymon did with toxins, for which reason the venoms thus depoisoned are called anavenins. Likewise, Esvold, making a very weak solution of the venom of Naja sputatrix and inactivating it, prepared a pharmaceutic product of definite properties known on the market as cobratoxin. Similarly, Brecher prepared a solution used in the treatment of trachoma and called trachozid. Still others have prepared extremely dilute solutions of venoms, without other modifications, and have used them for therapeutic purposes. It is well to remark here that the action of venoms varies much according to the dose. For instance, that of Bothrops jararaca, as von Klobusitzky found, has an anticoagulating effect in strong doses but is intensely coagulating in weak ones.

The venom of Naja sputatrix was recommended in the treatment of high retinal blood pressure, this indication being based on the pressure-

reducing effect acting, as it seems, directly on the walls of the vessels. Unfortunately, however, in opposition to the favorable results referred to by various authors are the observations of others who could not obtain similar results. The use of Naja venom was recommended for the treatment of glaucoma because of its miotic action and its effect in lowering ocular tonus, but it has not proved equally useful in the hands of other experimenters. For the relief of pain arising from inflammation of the cornea and of the uveal tract, or even due to increased ocular tonus, the use of a solution of the venom of Naja or Crotalus, applied by instillation or by subconjunctival injections, is based on the neurotoxic action of these venoms. In the treatment of trachoma the use of a derivative of the venom of Vipera berus and that of bees, known as trachozid, is advocated. This remedy, which has proved effective according to various experimenters, is applied in subconjunctival and intratarsal injections. But in spite of being unquestionably a useful element in the therapeutics of trachoma, it cannot as yet be considered as specific.

To avoid and combat bleeding during and after operations, and even in ocular disease in which bleeding is frequent, use has been made of derivatives of snake venom, with good results. The indication for this treatment is based on the coagulating action of these venoms, which seem to act as thrombin, transforming fibrinogen into fibrin.

Study of the various elements found in snake venoms, the action of which is not yet sufficiently known, will perhaps render possible in the future their more frequent use, and especially that of the principles which can be isolated from them. The venom, being composed of various elements acting differently, has had its therapeutic use limited by the impossibility of applying each pharmacodynamic property separately. The weakening of the venom by heat, by formaldehyde or by the neutralization of certain harmful elements, although it may have a selective action in the repression of certain undesirable elements, at the same time weakens the action which is to be utilized and retains, even though weakened, the undesirable elements.

Occurrence of Vertical Anomalies Associated with Convergent and Divergent Anomalies. Dr. James W. White and Dr. Harold W. Brown, New York.

This article will be published in full, with discussion, in a later issue of the Archives.

Transplantation (Implantation) of the Lacrimal Sac in Chronic Dacryocystitis. Dr. William H. Stokes, Omaha.

This article will be published in full, with discussion, in a later issue of the Archives.

Oculoglandular Diseases with Special Reference to Tularemia and Parinaud's Conjunctivitis. Dr. V. Reeves Hurst, Longview, Texas.

In 23 cases of oculoglandular disease, with histologic studies in 12, no clinical difference was noted between cases in which the leptothrix was found and cases in which it could not be demonstrated. Two cases of

oculoglandular tularemia were sharply differentiated from the cases of Parinaud's conjunctivitis by their clinical course and agglutination reaction, which bears out the contention that these are separate disease entities.

DISCUSSION

Dr. Sanford R. Gifford, Chicago: Dr. Hurst has had a remarkable experience in being able to see and diagnose 21 cases of Parinaud's conjunctivitis, and 2 cases of oculoglandular tularemia. Only one physician, Dr. Verhoeff, has had a greater experience with this form of conjunctivitis, and of the 45 patients from whom he examined material, I do not know how many he saw personally. About eight years ago I examined material from one of Dr. Hurst's patients and found what I considered elements of leptothrix in sections. They were few in number, however, and Dr. Verhoeff did not agree with my identification. In material from another patient, whom he saw not long after this, I was unable to find threads or other organisms. In material from 2 other patients which he sent this summer many undoubted leptothrix threads were present in the capillary spaces and free in the tissues. There can be no doubt, therefore, that in these 2 instances at least he was dealing with leptotrichosis conjunctivae.

In 18 of his cases the findings were negative. As I understand it, these cases were clinically similar to the ones in which leptotriches were found. Dr. Hurst states that in 12 of these cases tissue was not removed for sections, and it seems more than possible that leptotriches might have been found in some of these. In at least 1 of Dr. Hurst's cases in which I found many leptothrix threads, many giant cells were present, a finding which I believe differs from that of Dr. Verhoeff.

It still seems to me confusing and misleading to use the names tularemia and Parinaud's conjunctivitis interchangeably. Tularemia is clinically distinguishable from other diseases by the agglutination test, which is always positive in the second or third week, and usually by a history of contact with rabbits or squirrels. The systemic symptoms are much more marked than have generally been observed in cases described as Parinaud's conjunctivitis. No fatalities have been reported in the latter condition, and systemic symptoms are usually limited to slight fever and malaise for a few days after the onset.

The presence of eosinophilia, which was noted in a number of cases of leptotrichosis, is not constant enough to be of positive diagnostic value. Dr. Hurst found it in only half of his cases. Hence it may be considered suggestive but by no means essential to the diagnosis. I believe that it has not been reported in tularemia.

Ophthalmologists will continue to associate the name of Parinaud with the picture of unilateral adenopathy and conjunctival granulations. This is likely to be true in spite of modern refinements, which make possible a division of this picture into at least three etiologically distinct entities. Hence the suggestion which I proposed in 1934 still seems a reasonable one; that is, that the general picture be denoted as the conjunctivoglandular syndrome of Parinaud and that modern means be employed to differentiate the three diseases, which may in some cases somewhat resemble each other. These three conditions are, of course, leptotrichosis conjunctivae, the oculoglandular form of tularemia and conjunctival tuberculosis.

Book Reviews

Augenaerztliche Eingriffe. Eine Kurzgefasste Operationslehre. Edited by Prof. Dr. J. Meller, Chief of the First University Eye Clinic in Vienna. Fourth revised and enlarged edition, with contributions by J. Böck, K. Kofler, A. Pillat and L. Sallmann. Price, paper, 36 marks; cloth, 37.80 marks. Pp. 416, with 261 partly colored reproductions in the text and 2 plates. Vienna: Julius Springer, 1938.

This excellent textbook on ophthalmic surgery is well known to the ophthalmologic world through the three previous editions (the third in 1931) and the two English editions.

The advances and changes in ophthalmic technic during the past seven years have been reflected in this thoroughly revised and considerably enlarged fourth edition.

The operations discussed are those used, with few exceptions, in the First University Clinic in Vienna. They are presented in a clear, concise manner, and the text makes most agreeable reading.

Contributions by Böck, Kofler, Pillat and Sallmann have added new material in keeping with the high standard of this work. The book is divided into sixteen chapters.

The first chapter embraces probing of the lacrimal passages, operations on the canaliculi and extirpation of the tear sac. The latter operation is described in detail, with clear illustrative cuts, as in the previous editions.

External dacryocystorhinostomy and endonasal dacryocystorhinostomy as well as operations on the lacrimal gland are discussed in the second chapter. Special emphasis is placed on the Toti operation and the various steps described. Here, also, Prof. K. Kofler describes in a concise manner endonasal dacryocystorhinostomy; this section is well illustrated and comprehensively written. The chapter should be of special interest to the otolaryngologist.

The more familiar procedures for correcting spastic ectropion and senile ectropion are described in the third chapter.

The fourth chapter deals with ectropion resulting from scars, operations on the lids, restorations of the lid, correction of symblepharon, early plastic operations for burns and plastic operations on the orbit. The detailed descriptions and the many illustrations make the text clear and understandable.

Then follows a short chapter on operations for ectropion and trichiasis. Canthotomy, canthoplasty and tarsorrhaphy are discussed in the next chapter. The Fuchs and the Elschnig operations are described in detail.

The Eversbush, Hess and Motais operations are the procedures selected in the chapter dealing with ptosis.

The eighth chapter discusses squint. Tenotomy and advancement operations as performed in the Vienna Clinic are emphasized. A description of the cinch operation (R. O'Connor) is given, of which the author speaks favorably. The choice of operations and their relative

value, followed by a discussion on convergent and divergent squint, ends this chapter.

The ninth chapter considers enucleation, opticociliary neurotomy, exenteration of the orbital cavity and operations on the orbit. Meller favors the cartilage implant as recommended by Magitot after enucleation. A good description of the Krönlein operation is presented, with illustrations.

The next chapter, one of the high spots of this book, is a splendid treatise on cataract extraction, describing in detail the methods employed in extracapsular and intracapsular operations. Professor Meller still favors the extracapsular operation for surgeons with limited operative experience.

For intracapsular operations he uses the method of Knapp and Török or of Arruga rather than the Smith Indian method.

The eleventh chapter deals with discission and linear extraction.

Under the title "Operations for Reduction of Tension (Glaucoma)" the author deals with iridectomy, anterior sclerotomy and the trephine operations of Lagrange and Elliott. Dr. A. Pillat, of Graz, gives a good description of iridonclesis (Holth). His modifications include the conjunctival flap method of dissection; a smaller and more oblique scleral incision and the cutting of the iris meridionally but not to its base. Each pillar is then drawn into the angle of the scleral wound. Pillat claims that thus the iris is more securely held in place and that there is less tendency for it to slip back into the anterior chamber. Posterior sclerotomy, cyclodialysis (Heine) and other procedures are briefly mentioned. This chapter ends with the operations for secondary glaucoma.

Optical iridectomy, excision of a prolapsed iris, conjunctival plastic operations, operations to correct anterior synechiae and corneal transplantation are discussed in the thirteenth chapter. There is no mention made of the method for corneal transplantation so successfully done by Castroviejo.

The surgical treatment of retinal detachment under the heading of "Electrosurgical Treatment of Retinal Detachment" is edited by Dr. J. Blöck, of Vienna. Dr. Blöck has rewritten the previous text and given a lucid description of the various methods of electrocoagulation which are now generally used. The methods for localizing the tear are thoroughly considered.

A one point needle is favored, and the operation is performed under ophthalmoscopic control; it is a most valuable and often neglected procedure.

The undermining method of K. Linder is described by Privatdozent Dr. S. Sallmann, of Vienna. This is an elaborate procedure combining trephining and chemical cauterization (similar to Guist method) with an undermining of the tissues. It will probably never become popular but has been added to make the book more complete.

Extraction of intraocular foreign bodies is covered by the fifteenth chapter. In magnetic extractions Meller favors the anterior route.

The miscellaneous minor operations on the cornea, conjunctiva, lids and vitreous, such as periotomy and tattooing and operations for keratoconus, pterygium and chalazion, are treated in the last chapter.

The book ends with short paragraphs on the duties of the assistant and on the subject of local and general anesthesia.

This is an outstanding book; it is clear, concise and genuinely instructive, dealing with those operations used by the author, selected after years of exceptional experience and presented as only an able teacher is capable of doing. The reading will be a joy and of much profit to all German-reading ophthalmologists.

A translation into English of this new fourth German edition will be eagerly awaited and will be welcomed not only by the ophthalmologist but by all surgeons interested in ophthalmic surgery.

J. H. Ohly.

Refraction of the Eye. By Alfred Cowan, M.D. Price, $4.75. Pp. 319, including index, with 172 engravings and 3 colored plates. Philadelphia: Lea & Febiger, 1938.

The author has successfully accomplished his "desire to write a book on clinical refraction, employing the theory of ophthalmic optics in such a way that clinical aspects would emerge logically and in orderly sequence from their bases of scientific facts."

The part of the book dealing with the theory of ophthalmic optics is essentially a reprint of the author's book on this subject published in 1928, advantageously amplified in some respects. Retinoscopy and the use of crossed cylinders are much more completely described in the present volume. The author gives a satisfying discussion of the correction to be prescribed after the errors of refraction have been determined. Velonoskiascopy and the duochrome test are described.

Under "The Prescription" he discusses the types of lenses and of bifocal lenses to be prescribed. This is a particularly valuable feature of the book.

A few relatively unimportant criticisms may be made. The author still fails to discuss monocular diplopia and allied phenomena resulting from aberration of the refractive media. What is said in the page and one-half devoted to astigmatic charts is accurate but inadequate. He states that Lancaster uses a concave cylinder to produce an artificial astigmatism and implies that the purpose of this is to ascertain whether or not the patient is a "poor subject." The real purpose of this procedure should be to teach the patient what to observe.

The author discusses the question of visual efficiency in seven lines. Snell and Sterling's estimates are referred to, but the basis for these estimates is not given. As this question constantly arises in compensation cases, it might well have been more fully described.

About three fourths of a page is devoted to aniseikonia. It is stated that this, "according to Ames is defined as that condition of the eyes in which the size or shape of the ocular image (the impression that is carried to the brain) of one eye differs from that of the other." This is a highly inaccurate definition. A definition that aniseikonia is a condition in which the horopter is markedly abnormal would have been brief and correct. Determination of aniseikonia is simply determination of the horopter. No comment is made as to the reliability of the methods brought out at the Dartmouth Eye Institute for the determination of aniseikonia.

The author is confusing in his explanation relating to contact glasses. He states that the regularly curved portion of the glass is very thin and the refraction at its surfaces negligible! Yet he then states that the water between the contact glass and the cornea eliminates the corneal surface as a refracting surface. The truth of the matter is, of course, that the contact glass and the water behind it become the chief refractive media of the optical system.

The author does not discuss the relative merits of the various methods that he describes. Nor does he mention the routine methods found by him to be most useful in his thirty years' experience. Obviously all of the methods described need not be employed in every case, but the author gives no indications as to which may usually be omitted. He does not teach the beginner how to begin.

He makes no definite statement as to when cycloplegics should be used. He implies, however, that they should be used for all patients under 50 years of age and that each patient should be examined three times before the prescription is given. However, in an experience of about forty years, I have found that except in the case of young children cycloplegics are seldom needed and that usually a satisfactory prescription for glasses can be given after one examination.

I regard Dr. Cowan's book as of great practical value. There are few experienced ophthalmologists who would not profit by reading it, and every beginner should familiarize himself with its contents.

F. H. Verhoeff.

Principles and Practice of Perimetry. Dr. Luther C. Peter, Philadelphia. Fourth edition. Pp. 331, with 222 engravings and 5 colored plates. Philadelphia: Lea & Febiger, 1938.

When the third edition of this well known book appeared several years ago, it was suggested to the author that its value would be greatly enhanced if he had used a little more care in accurately describing the conditions under which each field was taken and in faithfully recording the findings. At least, the size and color of the test object used, the distance of the screen from the eye and the nature and intensity of the illumination should have been recorded for each field. Yet, by actual count, about 125 cuts, mostly of bilateral fields, taken without any of these necessary data, have been transferred from the second edition of this book, published in 1923, to this new edition.

In describing the methods of taking the fields, the author begins with what he calls the hand method. This is a crude modification of the well known confrontation method, which is useful and accurate when properly done. Instead of the hand method, the usual confrontation method should have been accurately described. Also, on page 87 the author again describes his well known hand campimeter. This description could well have been omitted, since this instrument can no longer be said to fill a useful place in the practice of perimetry.

Dr. Peter has included in this new edition a description of what Dr. John Evans calls "angioscotometry," which, according to Dr. Evans, means mapping out the perivascular lymph spaces, even including those around the small vessels, both central and peripheral. This is a

bold step on the part of Dr. Peter. He could well have waited for further confirmation of this claim of Dr. Evans by established authorities on perimetry, both in this country and abroad. It would have been well to have kept this matter in abeyance for a time.

With the exception of the comments on angioscometry, this new edition of Dr. Peter's well known book is practically identical with the previous edition.

A. H. THOMASSON.

Erbleiden des Auges, in Handbuch der Erbkrankeiten. Edited by Dr. Arthur Gütt, Berlin. Volume 5. Price, 24 marks. Pp. 310, with 212 illustrations. Leipzig: Georg Thieme, 1938.

Hereditary diseases is a subject which at present is occupying a great deal of attention in Germany, especially because of the movement to regulate these conditions by law. This book presents the present day knowledge of practically important hereditary diseases of the eye, with a plea for investigation and notification of all pertinent cases.

The subject is treated by eight German ophthalmologists under these headings: coloboma and deformities of the eyeball (Fleischer); glaucoma (Löhlein); corneal anomalies (Stock and Bücklers); hereditary cataract (Bücklers); albinism (Marchesani); color blindness (Marchesani); retinitis pigmentosa (Jess); macular degeneration (Jess); hereditary disease of the optic nerves (Wegner); refraction (Clausen), and strabismus (Harms).

Each subject is introduced by a clinical description with excellent illustrations, often in colors, then follow the form of hereditary transmission, with charts of family trees, and a consideration of the necessity or desirability for intervention in the endeavor to prevent its propagation. A bibliography concludes each chapter.

The ophthalmic reader will find this book an excellent introduction and book of reference for many conditions the importance of which from a genetic standpoint is becoming more and more realized.

ARNOLD KNAPP.

Zeitfragen der Augenheilkunde. Edited by Prof. W. Löhlein, Berlin. Price, 16.80 marks. Pp. 428, with 64 illustrations. Stuttgart: F. Enke, 1938.

Lectures on timely problems in ophthalmology were delivered in the postgraduate course in Berlin in 1938, under the leadership of Prof. W. Löhlein, the head of the University Eye Clinic in Berlin. The publication of these lectures will be appreciated by many who were unable to attend the course and will be of great value for future reference to the participants. In the selection of these courses topics not only of practical importance but of neighborhood subjects of more theoretic and fundamental importance were selected. A number of the lectures were devoted to neurologic subjects. The titles of the lectures were: "Pathology of Hereditary Diseases;" "The Dangers to the Eye During Pregnancy, Labor and Lactation," which were treated both by an oculist and by an obstetrican; "Problems Connected with the Lens;"

"Malignant Exophthalmos in Thyroid Disturbances;" "Lipoid Disturbances;" "Contact Glasses," and "Keratoplasty."

Professor Löhlein has made an excellent selection of timely topics and has selected an admirable group of speakers. The book will well repay careful study and frequent reference.

ARNOLD KNAPP.

Marihuana. By Dr. Robert P. Walton. Price, $3. Pp. 223. Philadelphia: J. B. Lippincott Company, 1938.

This book is a collection and summary of the literature concerning all phases of the marihuana drug habit. It deals with such topics as the history of the hashish vice, its distribution, its present status in the United States, the source of the plant, the technic of ingestion or administration, the hashish experience, acute and chronic effects and the chemistry and pharmacology of the drug Cannabis indica.

There are almost no points of particular ophthalmologic interest, though pupillary dilatation seems to be a fairly constant effect of acute intoxication by the drug.

O. P. PERKINS.

Zeitfragen der Augenheilkunde. Edited by Prof. W. Löhlein and Prof. W. Wegner. Second edition. Price, 8 marks. Pp. 346. Stuttgart: F. Enke, 1938.

A second edition of the lectures given in the postgraduate course in Freiburg in 1934 is now available. These lectures were reviewed in the February 1935 issue of the ARCHIVES (page 311).

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Bachler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, 81 Edmund St., Birmingham, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1.
Time: April 20-22, 1939.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Time: July 6-8, 1939.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung, China.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

Canadian Medical Association, Section on Ophthalmology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. Edward Jackson, 1008-A Republic Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 6:30 p. m., third Saturday of each month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT
President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA
President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY
Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. F. C. Smith, 106 W. 7th St., Charlotte.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Place: Statesville. Time: Sept. 21, 1939.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.
Place: Fargo. Time: May 1939.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St., N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606-20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Pl., Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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TREATMENT OF TRACHOMA WITH SULFANILAMIDE

POLK RICHARDS, M.D.
Medical Director in Charge of Trachoma Activities, Indian Service
ALBUQUERQUE, N. M.

WESLEY G. FORSTER, M.D.
Special Physician, Indian Service
FORT APACHE, ARIZ.

AND

PHILLIPS THYGESON, M.D.
NEW YORK

In February 1938 Dr. Fred Loe, of Rosebud, S. D., notified us of his successful treatment of trachoma in Indian subjects with sulfanilamide, the details of which he has recently published.[1] Favorable results were also reported by Lian.[2]

After obtaining marked improvement with this treatment in certain isolated cases and rapid cure in 2 cases of experimental trachoma in baboons, we decided to test the drug on a series of persons with previously untreated active follicular trachoma in whom corneal progress could be followed by means of the slit lamp and the corneal microscope.

Accordingly, we selected 14 Apache Indian children with active follicular trachoma, 12 to be treated and 2 to be kept untreated as controls. All children had pannus and exhibited corneal activity, as evidenced by dilated pannus vessels, subepithelial infiltrates and fluorescein-staining punctate epithelial lesions. Typical epithelial cell inclusions, characteristic of active trachoma, were demonstrated in epithelial scrapings from 13 of the 14 children. Sulfanilamide in a daily dose of ⅓ grain (0.0216 Gm.) per pound of body weight, with an equal amount of sodium bicarbonate, was given in four doses during the day. The results of treatment are summarized briefly in the accompanying table.

From the Division of Health, Office of Indian Affairs, Washington, D. C., in cooperation with the Department of Ophthalmology, College of Physicians and Surgeons, Columbia University, and the Institute of Ophthalmology, Presbyterian Hospital, New York.

1. Loe, F.: Sulfanilamide Treatment of Trachoma: Preliminary Report, J. A. M. A. **111**:1371 (Oct. 8) 1938.

2. Lian, B.: Sulfanilamide in the Treatment of Trachoma, Geneesk. tijdschr. v. Nederl.-Indië **78**:1058, 1938.

Summary of Results of Sulfanilamide Therapy in a Series of Trachomatous Subjects

No., Name	Age, Sex	Stage*	Intensity†	Bacteriologic Data	Inclusions	Cornea‡	Dose of Sulfanilamide	Progress Under Treatment	Final Result; 4½ Months
1 H. H.	12 F	IIa-III	+	Normal	+	4 degrees of pannus; Herbert's pits; infiltrates; epithelial lesions	⅓ grain per pound of body weight per day for 43 days	Cornea rapidly became inactive; lag in conjunctival improvement	Disease arrested
2 A. T.	8 F	IIa	++	Haemophilus influenzae Diplococcus pneumoniae	+	2 degrees of pannus; infiltrates; punctate epithelial lesions	Same as for H. H.	Rapid disappearance of corneal lesions	Disease arrested; slight conjunctival hyperemia
3 S. S.	13 F	IIb, O.D. IIb-III, O.S.	+++	Diplococcus pneumoniae	+	1 degree of pannus; infiltrates; punctate epithelial keratitis, slight in right eye and marked in left eye	Same as for H. H.§	Rapid disappearance of corneal lesions	Conjunctiva smooth; slight hyperemia; slight punctate epithelial keratitis
4 K. J.	13 F	I-IIa	+++	Diplococcus pneumoniae	+	1 degree of pannus; infiltrates; slight punctate epithelial keratitis	Same as for H. H.§	Rapid disappearance of corneal lesions; lag in disappearance of conjunctival follicles	Disease arrested; slight conjunctival hyperemia
5 E. T.	13 M	IIa-III	+	Diplococcus pneumoniae Haemophilus influenzae	+	2 degrees of pannus	Same as for H. H.§	Rapid corneal response	Disease arrested; slight conjunctival hyperemia
6 N. M.	9 M	IIa-III	+	Diplococcus pneumoniae	+	2 degrees of pannus in right eye, 1 degree in left eye; infiltrates; punctate epithelial keratitis	Same as for H. H. for 41 days§	Rapid corneal response; delayed conjunctival response	Conjunctiva smooth; cornea of right eye inactive; one infiltrate in cornea of left eye
7 L. W.	10 M	I-IIa	++	Normal	+	1 degree of pannus; infiltrates; epithelial lesions	Same as for H. H. for 45 days§	Rapid corneal response; delayed conjunctival response	Conjunctiva smooth; slight hyperemia; cornea healed in right eye; slight punctate epithelial keratitis in left eye
8 L. M.	15 M	IIa-III	+	Diplococcus pneumoniae	—	2 degrees of pannus in right eye, 3 degrees in left eye; Herbert's pits; scars; infiltrates; epithelial lesions	Same as for H. H. for 41 days§	Rapid corneal improvement	Disease healed in right eye; slight conjunctival hyperemia and slight epithelial keratitis in left eye
9 R. S.	10 F	IIa	++	Normal	+	2 degrees of pannus in right eye, 1 degree in left eye; infiltrates; punctate epithelial keratitis	Same as for H. H. for 10 days§	....	Conjunctiva healed; slight punctate epithelial lesions
10 A. T.	11 F	IIb-III	++	Haemophilus influenzae Diplococcus pneumoniae	+	3 degrees of pannus in right eye, 4 degrees in left eye; pits; infiltrates; epithelial lesions	Same as for H. H. for 19 days	Rapid corneal response; delayed conjunctival response	Disease arrested; slight conjunctival hyperemia
11 E. E.	12 F	IIa-III	± O.D. ++ O.S.	Diplococcus pneumoniae	+	2 degrees of pannus; ulcer in left eye; infiltrates; epithelial lesions	Same as for H. H. for 53 days	Rapid corneal response	Disease arrested in left eye; slight punctate keratitis in right eye
12 R. B.	12 M	I Unilateral	++	Normal	+	1 degree of pannus; infiltrates; epithelial lesions	Same as for H. H.	Rapid corneal and conjunctival response; inactive 53 days	Disease arrested
Controls									
13 J. M. J.	8 M	IIa	+++	Normal	+	2 degrees of pannus; infiltrates; epithelial lesions	None	No change	No change
14 T. J.	9 F	IIa	++	Normal	+	1 degree of pannus; limbal follicles; infiltrates; epithelial lesions	None	No change	No change

* The stage of trachoma is given according to MacCallan's classification: I, incipient; IIa, follicular hypertrophy predominant; IIb, papillary hypertrophy predominant; III, cicatrices formed.

† The intensity of trachoma was estimated + to ++++.

‡ Pannus was measured in four degrees, the first degree being incipient pannus and the fourth degree, complete pannus.

§ The dose of sulfanilamide was increased to ½ grain per pound of body weight on the seventh day of treatment.

Striking improvement was manifest in all 12 of the treated children. The appearance of the cornea improved during the first week of treatment and was characterized by the disappearance of subepithelial infiltrations, a reduction in the caliber of the vessels of the pannus and a diminution in the number of fluorescein-staining epithelial lesions. Conjunctival improvement first became noticeable about the end of the second week of treatment and was characterized by a diminution in conjunctival secretion and a thinning and paling of the membrane. There was, however, a decided lag in the disappearance of follicles; only 1 child (no. 12) showed a normal conjunctiva at the end of the period of treatment, and in this instance the trachoma was in a very early stage. But at the time of the final examination, four and one-half months after the beginning of treatment, the conjunctivas of all 12 children were smooth and follicle free; slight hyperemia persisted in 7. At this time corneal examination with the slit lamp showed a disappearance of infiltrates in all but 1 of the 24 eyes; this eye showed a single infiltrate remaining, near the upper limbus. Noteworthy was the disappearance of epithelial lesions in all but 7 of the 24 eyes. In only 1 of the 7 were the lesions as numerous as they had been before treatment.

The 2 controls showed no definite change in the intensity of their conjunctival or corneal trachoma during the period of observation. Both were then placed on ½ grain (0.0324 Gm.) of sulfanilamide per pound of body weight per day. After three weeks the trachoma of 1 control (no. 14) had become inactive; the other (no. 13) showed considerable improvement.

The disappearance of the trachoma virus from the conjunctivas of the children treated with sulfanilamide was indicated by our repeated failure to demonstrate inclusion bodies during the period of treatment and by the failure of pooled epithelial scrapings, taken the twenty-ninth day, to infect baboons. The 2 controls, on the other hand, showed inclusion bodies when tested at weekly intervals.

No indications of toxicity other than mild headache was observed as a result of sulfanilamide therapy. No leukopenia occurred in any patient.

It is of interest that pneumococci or influenza bacilli or both were demonstrated in cultures from the conjunctivas of 8 of the 12 children. After thirty days of sulfanilamide therapy the conjunctival flora of only 3 of the 8 was normal. There was no difference in the rate of clinical improvement of the trachoma in these children and in those in whom the bacterial flora was unaffected.

COMMENT

The therapeutic effect of sulfanilamide in this series of patients was beyond question. On the basis of the appearance of the conjunctivas

alone the disease would be considered inactive in all 12 children. However, the presence of a single subepithelial infiltrate in 1 eye would seem to indicate continuing corneal activity in this instance. The significance of continued epithelial lesions without subepithelial infiltrates in 6 other eyes is difficult to evaluate, but it may be that they represent minimal trachomatous activity. In any event, sulfanilamide therapy in this series of cases proved far more rapid and effective than any other type of antitrachomatous therapy so far employed by us.

CONCLUSION

A series of 12 Indian children, all showing active trachoma with follicular hypertrophy and pannus, were treated with sulfanilamide. All showed striking improvement, and at the end of four and a half months the conjunctiva in each instance had become follicle-free and smooth. Except in 1 eye, there was a disappearance of corneal infiltrates and an apparent arrest of corneal activity. Untreated children used as controls showed no improvement during the period of observation but improved rapidly when sulfanilamide therapy was instituted. Sulfanilamide therapy caused a disappearance of the epithelial cell inclusion bodies characteristic of active trachoma.

EFFECT OF CERTAIN CHEMICAL STIMULI ON THE CALIBER OF THE RETINAL BLOOD VESSELS

IRVING PUNTENNEY, M.D.

CHICAGO

It is a matter of considerable importance to inquire into the physiologic and pharmacologic reactions of the retinal blood vessels. As Mylius [1] pointed out, these reactions are complicated, and the correlation of certain experimental findings with clinical therapy depends on a clear understanding of these responses.

The primary concern of this study of the retinal blood vessels is with the underlying forces which produce vasodilatation. This assumes therapeutic importance, as the treatment for spasm [2] and closure of the central blood vessels relates to measures which will produce immediate vasodilatation. Of the chemical agents available, acetylcholine and the nitrites have been the drugs most frequently employed. In general, these drugs produce marked relaxation of the vascular musculature with an associated drop in the systemic blood pressure.

The favorable influence of these therapeutic measures has been recorded repeatedly in the literature. Many of these accounts appear convincing, but within recent years certain discrepancies have found their way into the literature, and the observations have not always been in accord.

In an attempt to evaluate these findings it becomes expedient to rely on records which have been made by methods more accurate than ordinary ophthalmoscopic observation. This, in itself, is not an attempt to contradict personal observation but rather an endeavor to adopt a more accurate and scientific method of approach.

There are two comparatively accurate means at one's disposal for measuring the retinal blood vessels. The first, the micrometer method, gives readings which may be compared with estimates made at a later date. The second, the photographic method, is by far the more precise.

From the Department of Ophthalmology, Northwestern University Medical School.

This work was made possible in part by a grant from the Council of Physical Therapy of the American Medical Association and in part by a grant from the John and Mary Markle Foundation.

1. Mylius, K.: Funktionelle Veränderungen am Gefäss-system der Netzhaut, Berlin, S. Karger, 1928.

2. Sedan, J., and Jayle, G. E.: Ann. d'ocul. **73**:607, 1936; **74**:6, 1937.

It is possible by this procedure to secure photomicrographs by direct microscopy; these records offer indisputable evidence of the results which occurred.

METHODS FOR MEASURING THE CALIBER OF THE RETINAL BLOOD VESSELS

All photographic procedures fall into two classes. The first is the simplest and is exemplified by the Nordenson camera.[3] This apparatus, which utilizes Gullstrand's method of reflex-free central ophthalmoscopy, is of great value in producing documentary evidence of retinal changes. The magnification, however, is limited to approximately six times, and this places a limitation on its accuracy in calibrating the retinal vessels. Despite this disadvantage, this method is the one more frequently employed clinically. In some cases it is advantageous to incorporate a micrometer scale in the picture. Simon and Goldstein [4] have described a method for doing this.

The second procedure entails the use of a planoconcave contact glass for neutralizing the optical system of the eye. This glass was first described by Koeppe [5] in 1918 and recommended for the purpose of examining the vitreous and the fundus with the slit lamp.[6] Lambert [7] in 1934 adopted this glass as an auxiliary in a new method for direct microscopy of the living eye. The mechanism, which is the only good experimental means of photographing the retinal vessels in animals, consists of a standard Leitz Ultrapak vertical illuminator mounted on an ordinary microscope barrel (fig. 1). The photographs are made by replacing the standard ocular with a miniature camera (Leica) equipped with a Leitz Micro-Ibsor lateral view finder and micrometer ocular attachment. The contact glass permits direct focusing on the retinal blood vessels, and the manipulation of this glass is facilitated by mounting it on a clear, clean glass slide. As Lambert has pointed out, the bubbles must be removed from the space between the contact glass and the cornea by the instillation of light liquid petrolatum. It is absolutely essential that all of the air be removed, since the presence of even one small bubble can completely distort the optical system and render the photographs inaccurate.

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3. Nordenson, J. W.: Hygiea **77**:1538, 1915; **87**:586, 1925. Hartinger, H.: Klin. Monatsbl. f. Augenh. **83**:579, 1929.

4. Simon, C., and Goldstein, I.: New York State J. Med. **35**:901, 1935.

5. Koeppe, L.: Arch. f. Ophth. **95**:282, 1918; **96**:232, 1918.

6. Meesmann, A.: Klin. Monatsbl. f. Augenh. **66**:417, 1921. Koeppe, L.: Arch. f. Ophth. **109**:454, 1922.

7. Lambert, R. K.: A Method for the Study of the Retinal Circulation, Arch. Ophth. **12**:868 (Dec.) 1934.

The actual technic of photography is not difficult after the contact glass is in place. The image is focused by slowly lowering the tube, and the sharp focus of the image in the viewing telescope corresponds to a similar focus on the photographic plate. The lateral view finder permits observation during the exposure.

The nonphotographic procedures depend on estimating the caliber of the retinal blood vessels by comparing them with a standard graticule scale or by utilizing either a split prism or a micrometer ocular. The graticule was first described by Neame [8] in 1936. It is advocated for use with the ophthalmoscope, and vessels as small as 0.075 mm. can be compared on the projected scale. The split prism ocular was first adapted to the slit lamp by Goldmann [9] and introduced into ophthalmology by Hendriksson [10] in 1924. This device gives a means of comparing the caliber of the vessels with the diameter of the disk. In the hands of Lobeck [11] it appears to be more accurate than the direct reading scale of the micrometer ocular.

REVIEW OF LITERATURE ON THE REACTIONS OF THE RETINAL BLOOD VESSELS

The methods which have been used to alter the caliber of the retinal blood vessels fall into two classes: chemical and physical. In this work an attempt has been made to investigate experimentally the nature of these reactions by the administration of certain drugs and to present the proof in the form of photomicrographs taken of the vessels in the living animal.

Epinephrine.—Epinephrine is a powerful sympathomimetic drug. It produces vasoconstriction by peripheral stimulation of the smooth muscle in the arteries, veins and capillaries. This response is relatively greater in the arterioles because of the abundance of smooth muscle. It has been shown that the constricting action on the pulmonary [12] and coronary [13] arteries is weak and that in the living animal these vessels usually dilate passively with the increased blood pressure. Forbes,[14] Wolff [15] and

8. Neame, H.: Tr. Ophth. Soc. U. Kingdom **56**:155, 1936.
9. Goldmann: Ztschr. f. Augenh. **78**:89, 1932.
10. Hendricksson, V.: Hygiea **86**:887, 1924.
11. Lobeck, E.: Arch. f. Ophth. **133**:152, 1935; **136**:439, 1937.
12. Ward, H. C., Jr.: J. Exper. Med. **14**:326, 1911.
13. Brodie, T. G., and Cullis, W. C.: J. Physiol. **43**:313, 1911.
14. (*a*) Forbes, H. S.; Finley, K. H., and Nason, G. I.: Cerebral Circulation: XXIV. A. Action of Epinephrine on Pial Vessels; B. Action of Pituitary and Pitressin on Pial Vessels; C. Vasomotor Response in the Pia and in the Skin, Arch. Neurol. & Psychiat. **30**:957 (Nov.) 1933. (*b*) Forbes, H. S., and Wolff, H. G.: III. Vasomotor Control of the Cerebral Vessels, ibid. **19**:1057 (June) 1928.
15. (*a*) Wolff, H. G.: Physiol. Rev. **16**:545, 1936. (*b*) Forbes and Wolff.[14b]

others studied the effect on the cerebral vessels. It has been generally observed that vasoconstriction occurs after epinephrine has been applied locally, and when the drug is administered intravenously the local constrictor effect is overcome and the cerebral vessels subsequently dilate. Wolff stated: "The effect of any given amount of epinephrine is the resultant of two opposing forces—a weak local constrictor action and the relatively stronger expansile action of the raised systemic arterial pressure."

The effect of epinephrine on the retinal blood vessels has frequently been reported. Hirschfelder [16] studied the retinal vessels of cats and rabbits and reported vasoconstriction with the use of epinephrine. Cohen and Bothman [17] observed rabbits ophthalmoscopically and noted that "adrenalin, administered intravenously, produced a temporary constriction of the arteries with a flushing of the veins from the beginning." Agatston [18] in 1933 reported producing spasm in the arteries of rabbits by injecting the drug; he was convinced that epinephrine is an active agent in the production of pathologic spasm.

Bailliart [19] attacked the problem in an ingenious manner. He trephined the sclera and then applied epinephrine locally through the trephine opening. Prolonged vasoconstriction occurred in the treated area.

Lambert [20] in 1935 reported the first photomicrographic studies on the effect of epinephrine in cats. He employed the technic previously described and injected the drug intravenously in various sized doses; vasodilatation occurred in a large percentage of the cases. Lambert accounted for this increase in the caliber of the vessels on the basis of a passive dilatation secondary to an increase in arterial tension.

Riser, Couadau and Mériel [21] adopted Lambert's technic in 1936, and their findings corroborate the work of both Lambert and Bailliart. In a series of 30 cats, injection of epinephrine into the carotid artery produced dilatation of the retinal vessels amounting to an increase of from 15 to 20 per cent in caliber. In a second series of 65 cats, intravenous (femoral) injections of epinephrine produced an increase of from 15 to 20 per cent in the caliber of the vessels in practically all of the animals. Constriction of the arteries was observed only when the drug was applied locally through a scleral trephine opening.

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16. Hirschfelder, A. D.: J. Pharmacol. & Exper. Therap. **6**:597, 1915.
17. Cohen, S. J., and Bothman, L.: Am. J. Physiol. **81**:665, 1927.
18. Agatston, S. A.: Am. J. Ophth. **16**:327, 1933.
19. Bailliart, M. P.: Tunisie méd. **29**:51, 1935.
20. Lambert, L.: Am. J. Ophth. **18**:1003, 1935.
21. Riser, Couadau and Mériel: Presse méd. **44**:1225, 1936.

Nitrites.—Amyl nitrite was first introduced into medicine by Brunton [22] in 1867 and recommended for the treatment of angina pectoris. This drug, together with other members of its group, produces vasodilatation by directly lessening the tone of vascular musculature. The reaction occurs peripherally, and dilatation of the coronary [23] and cerebral [24] vessels takes place in spite of the decrease in arterial tension.

Story [25] in 1899 was apparently the first ophthalmologist to describe the successful use of amyl nitrite in the treatment of embolism of the central artery of the retina. Similar successes were reported by Jennings [26] in 1911 and by Hird [27] in 1912. A more significant contribution is that reported by Imre.[28] He employed the drug in the treatment of certain chronic diseases of the fundus and explained his favorable results on the basis of vasodilatation of the retinal vessels. Procksch,[29] Deutschmann,[30] Genet [31] and others have been equally impressed with their results.

Lobeck,[11] a German worker, made use of the split prism ocular of Hendriksson and estimated the caliber of the vessels in a small series of cases. He recorded a slight increase in the diameter of the arteries and veins after the inhalation of amyl nitrite.

In 1931 Haessler and Squier [32] reported a careful piece of work in which the clinical effects of amyl nitrite were recorded with the Nordenson camera. The authors were unable to discern any relation between the caliber of the retinal vessels after the inhalation of amyl nitrite and the systolic blood pressure before and after the inhalation of the drug. King [33] has also observed no change in the caliber of the vessels after the inhalation of this chemical.

Lambert, in an article previously referred to, published the most convincing proof of the nature of the reaction to nitrites in cats. In a series of 6 animals there was a narrowing of the vessels, with changes

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22. Brunton, T. L.: Lancet **2**:97, 1867.

23. Loeb, V.: Arch. f. exper. Path. u. Pharmakol. **51**:64, 1903.

24. (*a*) Leake, C. D.; Loevenhart, A. S., and Muehlberger, C. W.: Dilatation of Cerebral Blood Vessels as a Factor in Headache, J. A. M. A. **88**:1076 (April 2) 1927. (*b*) Florey, H. W.: Brain **48**:43, 1925. (*c*) Wolff.[15a] (*d*) Schneider, M., and Schneider, D.: Arch. f. exper. Path. u. Pharmakol. **175**:640, 1934.

25. Story, J. B.: Tr. Ophth. Soc. U. Kingdom **19**:87, 1899.

26. Jennings, R. E.: Ann. Ophth. **20**:707, 1911.

27. Hird, R. B.: Ophthalmoscope **10**:370, 1912.

28. Imre, J., abstracted, Klin. Monatsbl. f. Augenh. **85**:128, 1930.

29. Procksch, M.: Klin. Monatsbl. f. Augenh. **85**:795, 1930.

30. Deutschmann, R.: Klin. Monatsbl. f. Augenh. **88**:168, 1932.

31. Genet, L.: Bull. Soc. d'opht. de Paris, May 1932, p. 329.

32. Haessler, F. H., and Squier, L. T.: Tr. Am. Ophth. Soc. **29**:254, 1931.

33. King, G. L., Jr.: Am. J. Ophth. **12**:827, 1929.

proportional to the drop in blood pressure in all save 1 animal. These changes were all recorded photographically.

Cohen and Bothman[17] described the action of glyceryl trinitrate administered intravenously to rabbits. In their series dilatation of the arteries was observed, with no change in the caliber of the veins.

Esterman,[34] Duggan,[35] Pflimlin,[36] Jaensch[37] and other clinicians have also reported favorably on the use of sodium nitrite. Esterman in a recent article stated: "The blood pressure dropped to 80/60, the retinal arteries increased to almost twice their normal size, and the obstruction disappeared within a short time."

Acetylcholine and Choline Derivatives.—Another drug which has been used extensively during the last few years is acetylcholine. It produces symptoms similar to those produced by stimulation of the parasympathetic nervous system. Studies have shown that in addition to this reaction the drug exerts a powerful vasodilator effect on the blood vessels which have no known parasympathetic innervation. It has been suggested that this chemical may be similar to the intermediate substance produced by stimulation of the parasympathetic nervous system.

Pharmacologically, the reaction of acetylcholine may be divided into two types: (1) parasympathetic, or muscarine, action, which is characterized by slowing of the heart, vasodilatation and a decrease in the level of the general blood pressure; (2) sympathetic, or nicotine, action, which results in an elevation of the blood pressure. This combination of reactions was first observed by Dale[38] in 1914; he emphasized that the muscarine activity was by far the more conspicuous. Four years later Fuhner[39] reported that small doses of physostigmine greatly increased the degree of stimulation of the parasympathetic nervous system. This observation has since been corroborated by Loewi and Navratil[40] and others, who have suggested that physostigmine probably inhibits the effect of tissue esterase on the acetylcholine. Loewi also reported abolishing this synergy with suitable doses of atropine. Feldberg and Mintz[41] have more recently confirmed this finding.

Acetylcholine was first introduced into medicine by Villaret and Justin-Besançon[42] in 1926. These clinicians claimed considerable suc-

34. Esterman, B.: New York State J. Med. **37**:296, 1937.
35. Duggan, W. F.: Vascular Basis of Tobacco Amblyopia: Treatment with Nitroscleran, Arch. Ophth. **13**:1059 (June) 1935.
36. Pflimlin, R.: Klin. Monatsbl. f. Augenh. **85**:287, 1930.
37. Jaensch, K.: Klin. Monatsbl. f. Augenh. **77**:189, 1926.
38. Dale, H. H.: J. Pharmacol. & Exper. Therap. **6**:147, 1914.
39. Fuhner, H.: Arch. f. exper. Path. u. Pharmakol. **82**:51, 1918.
40. Loewi, O., and Navratil, E.: Arch. f. d. ges. Physiol. **219**:689, 1926.
41. Feldberg, W., and Mintz, B.: Arch. f. d. ges. Physiol. **233**:657, 1933.
42. Villaret, M., and Justin-Besançon, L.: Bull. et mém. Soc. méd. d. hôp. de Paris **50**:465, 1926.

cess with this drug in the treatment of Raynaud's disease; the clinical improvement was attributed to vasodilatation.

It is generally accepted that acetylcholine dilates the cerebral vessels. Wolff [24c] found pial dilatation averaging 22 per cent in spite of an average fall in blood pressure of 69 per cent.

In the field of ophthalmology the French physicians have been the most enthusiastic. Kalt [43] in 1928 reported the first successful use of the drug in the treatment of a patient with occlusion of the central retinal artery. A more significant contribution, however, is that of Villaret, Schiff-Wertheimer and Besançon.[44] In a series of 25 clinical cases, acetylcholine was injected subconjunctivally, and their results are quoted as follows: "Dilatation of the arteries was found positive in eleven (11), five (5) more were uncertain and nine (9) were negative." Orr and Young [45] injected 8 minims (0.49 cc.) of the drug subconjunctivally and found that this quantity was sufficiently large to cause dilatation of the vessels in their case of occlusion of the central retinal artery.

Redslob [46] applied the principle of Bailliart and injected acetycholine directly into the vitreous of a patient suffering from spasm of the central retinal artery. The spasm disappeared within a short time, and no ill effects were observed following the use of this direct method of treatment. Other noteworthy contributions are those of Bailliart,[47] Villard, Dejean and Temple,[48] Rochon-Duvigneaud,[49] Gallois,[50] de Saint-Martin [51] and de Vaucleroy.[52] More recently, Parsons, McMullen and Juler [53] reported negative results with the drug in the treatment of occlusion of the central retinal vessels.

Extensive pharmacologic studies have also been made with many of the other choline derivatives. Hunt and Taveau [54] in 1911 synthesized a

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43. Kalt, M.: Bull. Soc. d'opht. de Paris, July 1928, p. 309.
44. Villaret, M.; Schiff-Wertheimer, and Justin-Besançon, L.: Compt. rend. Soc. de biol. **98**:909, 1928.
45. Orr, H. C., and Young, H. J.: Brit. M. J. **1**:1119, 1935.
46. Redslob, E.: Bull. Soc. d'opht. de Paris, March 1930, p. 143.
47. Bailliart, P.: Bull. Soc. d'opht. de Paris, June 1928, p. 245; Arch. d'opht. **46**:172, 1929.
48. Villard, H.; Dejean, C., and Temple, J.: Arch. d'opht. **49**:72, 1932.
49. Rochon-Duvigneaud: Arch. d'opht. **48**:143, 1931.
50. Gallois, J.: Bull. Soc. d'opht. de Paris, June 1931, p. 298.
51. de Saint-Martin: Ann. d'ocul. **168**:102, 1931.
52. de Vaucleroy: Arch. d'opht. **51**:116, 1934.
53. Parsons, J.: Proc. Roy. Soc. Med. **30**:300, 1937. McMullen, W. H.: ibid. **30**:300, 1937. Juler, F.: ibid. **30**:300, 1937.
54. Hunt, R., and Taveau, R. De M.: Effects of a Number of Derivatives of Choline and Analogous Compounds on Blood-Pressure, Hygienic Laboratory Bulletin 73, United States Public Health Service, 1911.

large number of these closely related compounds, and of this group acetylbetamethylcholine chloride (mecholyl) appeared to be the most promising. Menge [55] experimented with this chemical and compared its rate of disintegration in the tissues with that of acetylcholine. This work indicated that mecholyl was far more resistant to tissue hydrolysis. Various values have been given as to the comparative strength of the two drugs. Weiss and Ellis [56] have found mecholyl to be two hundred times as powerful in man; this value is probably too high. Hunt and Renshaw [57] considered the actions of the drug to be limited almost entirely to stimulation of the parasympathetic nervous system. This finding has been corroborated by Simonart [58] and others.

Mecholyl has not been used extensively in the field of ophthalmology, although numerous references are made to its miotic action.[59] Recently Myerson and Thau [60] reported dilatation of the retinal arteries after the instillation of a 1 to 10 per cent aqueous solution of the chemical. Photographic evidence is lacking.

The two other choline derivatives which should be mentioned are carbaminoylcholine (doryl) and the ethyl ester of betamethylcholine. Both of these drugs, though less potent than mecholyl, are powerful stimulants of the parasympathetic nervous system, and when they are instilled into the conjunctival sac miosis occurs.[61] There are no references in the literature concerning their effect on the retinal blood vessels.

EXPERIMENTAL STUDIES

Methods and Procedure.—The technic adopted for these studies was the same as that described by Lambert in 1934. The camera and vertical illuminator were mounted directly on a microscope barrel and attached to an upright stand, as shown in figure 1. The head rest was made with a strip of metal and secured to the base of the upright stand with a movable clamp. In order to secure uniform pictures, it was necessary to hold the contact glass with a clamp so that the under surface of the objective was parallel with it. After the concave surface

55. Menge, G. A.: J. Biol. Chem. **10**:399, 1911; **13**:97, 1912; in Hunt and Taveau.[54]

56. Weiss, S., and Ellis, L. B.: J. Pharmacol. & Exper. Therap. **52**:113, 1934.

57. Hunt, R., and Renshaw, R. R.: J. Pharmacol. & Exper. Therap. **51**:237, 1934.

58. Simonart, A.: J. Pharmacol. & Exper. Therap. **46**:157, 1932.

59. Villaret, M.; Justin-Besançon, L.; Cachera, R., and Said: Ann. de méd. **22**:385, 1932; cited in Methyl-Acetyl-Choline, Annotations, Lancet **1**:812, 1933. Hunt, R.: J. Pharmacol. & Exper. Therap. **55**:268, 1935.

60. Myerson, A., and Thau, W.: Human Autonomic Pharmacology: Effect of Cholinergic and Adrenergic Drugs on the Eye, Arch. Ophth. **18**:78 (July) 1937.

61. Applemann, M.: Compt. rend. Soc. de biol. **115**:1359, 1934. Velhagen, K., Jr.: Arch. f. Augenh. **107**:319, 1933. Comroe, J. H., Jr., and Starr, I., Jr.: J. Pharmacol. & Exper. Therap. **49**:283, 1933.

of the contact glass was filled with light liquid petrolatum and all of the bubbles were displaced, this clamp was fastened to the nearby stand. Eastman super X film was used exclusively, and all of the exposures were made at one fifth of a second with the bulb overloaded. An attempt was made to snap the pictures only at the end of expiration, as this reduced respiratory interference to a minimum. Edwald no. 12 was found to be a satisfactory developer.

Dogs were used as experimental animals and kept under deep anesthesia with intraperitoneal or intravenous injections of pentobarbital sodium. It was found absolutely necessary to select dogs with light colored fundi, weighing between 10 and 14 Kg. Simultaneous blood pressure tracings were made during some of the experiments. Canthotomy was performed on all of the animals, and bleeding was controlled with the cautery. Medication was administered subconjunctivally, retrobulbarly, subcutaneously and intravenously.

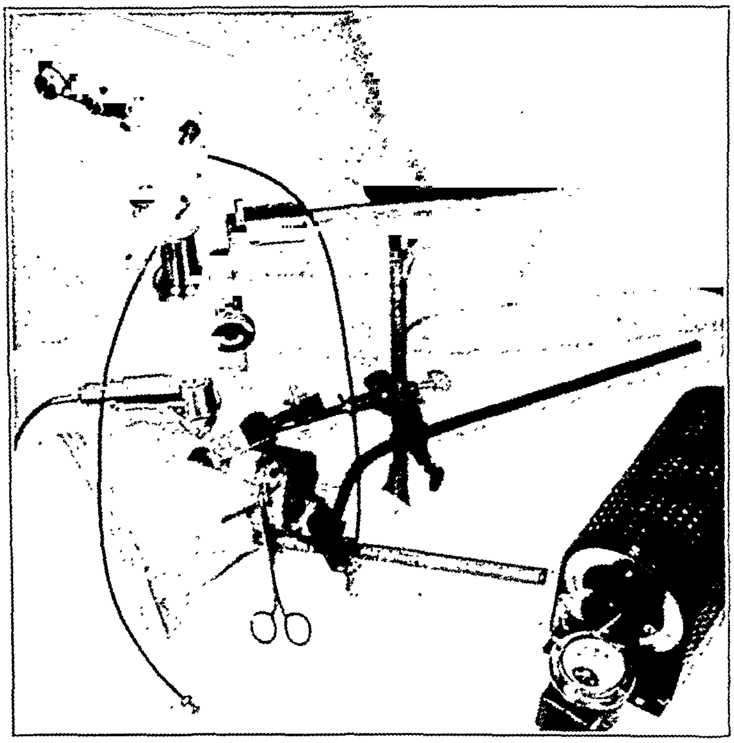

Fig. 1.—Instrument for photographing the retinal blood vessels in living animals.

RESULTS

Epinephrine.—In this part of the experimental work epinephrine hydrochloride was administered to 16 dogs (table 1). Ten animals received the medication by injection into the femoral vein, 4 by subcutaneous injection and 2 by retrobulbar injection. In 2 experiments there was a questionable increase in the caliber of the arteries (fig. 2), and in 7 the veins dilated. These reactions closely paralleled the change in blood pressure and lasted approximately three minutes. In order to study the effect of a decrease in the intraocular tension, paracentesis was performed on 2 of the dogs (fig. 3) prior to the intravenous injection

of epinephrine. Neither vasodilatation nor constriction occurred, either before or after the administration of the drug.

The retinal vessels were observed throughout each experiment by means of the viewing side arm on the Micro-Ibsor attachment. Careful records of ophthalmoscopic observation were made during this time and saved for comparison with the photographic negative. These personal observations were usually found to be inaccurate.

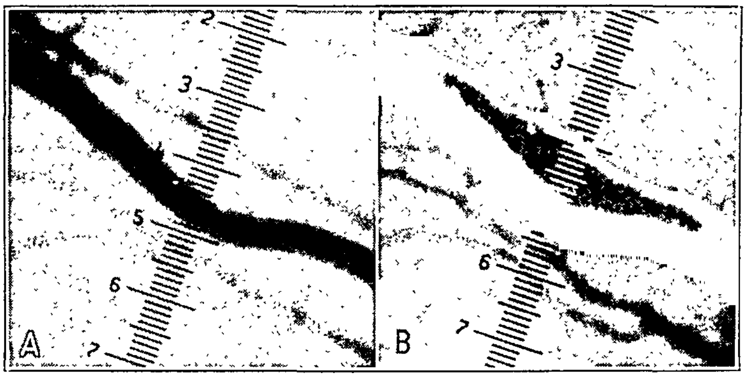

Fig. 2 (animal 30).—The effect of an intravenous injection of epinephrine on the caliber of the retinal vessels. *A*, before injection; *B*, after injection.

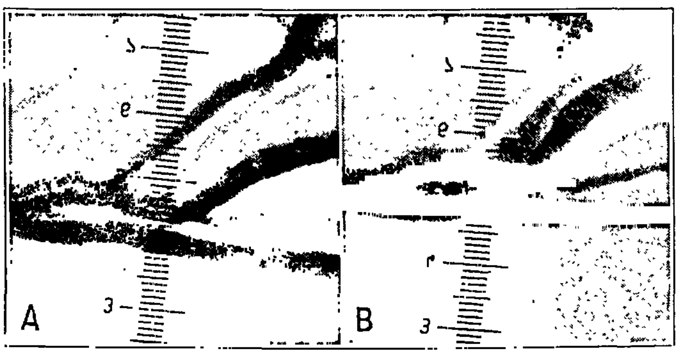

Fig. 3.—The effect of removing 3 minims of aqueous from the anterior chamber. *A*, before removal; *B*, after removal.

Nitrites.—Only 7 animals were tested in this group. Four received sodium nitrite by vein, 2 were given inhalations of amyl nitrite and 1 received glyceryl trinitrate by vein (table 2). All of these experiments gave negative results.

Mecholyl and Ethyl Ester of Betamethylcholine.—In this group mecholyl was administered to 20 dogs. Mecholyl chloride was administered to 13 animals by injections into the femoral vein, to 1 by subcutaneous injection and to 1 by retrobulbar injection; 5 were given

TABLE 1.—*Effect of Epinephrine on the Caliber of the Retinal Vessels*

Animal No.	Quantity of Epinephrine Hydrochloride Administered	Caliber of Vessels in Terms of Micrometer Readings: Before Medication		After Medication		Length of Time Observed, Minutes	Systemic Reaction
		Artery	Vein	Artery	Vein		
1	0.5 cc. of 1:10,000 sol. intravenously	2.0	4.0	2.0	4.0	20	Mild
3	1 cc. of 1:10,000 sol. intravenously	2.5	4.5	2.5	4.5	25	Mild
4	1 cc. of 1:1,000 sol. intravenously	2.0	4.0	2.0	5.0	20	Marked
5	1 cc. of 1:1,000 sol. intravenously	2.0	4.0	2.0	4.0	20	Marked
8	0.75 cc. of 1:1,000 sol. intravenously	2.0	3.5	2.0	4.0	20	Marked
9	0.75 cc. of 1:1,000 sol. intravenously	2.0	3.5	2.0	4.5	20	Marked
29	1 cc. of 1:1,000 sol. intravenously	2.5	5.0	2.5	6.0	20	Marked
30	1 cc. of 1:1,000 sol. intravenously	2.0	5.0	2.0	6.0	10	Marked
53	Paracentesis*	1.5	4.0	1.5	4.0	4	
	0.75 cc. of 1:1,000 sol. intravenously	1.5	4.0	1.5	4.0	35	Moderate
10	0.5 cc. of 1:1,000 sol. subconjunctivally	2.5	4.5	2.5	4.5	25	Mild
28	1 cc. of 1:1,000 sol. subcutaneously	2.5	5.0	2.5	5.5	30	Mild
31	1 cc. of 1:1,000 sol. subcutaneously	2.0	4.5	2.0	4.5	25	Mild
32	1.5 cc. of 1:1,000 sol. subcutaneously	2.0	4.5	2.0	4.5	30	Moderate
17	1 cc. of 1:1,000 sol. retrobulbarly	1.5	4.5	1.5	4.5	20	Moderate
27	0.7 cc. of 1:10,000 sol. retrobulbarly	2.5	4.5	2.5	5.0	25	Moderate
49	Paracentesis*	1.5	6.0	1.5	6.0	4	
	1 cc. of 1:1,000 sol. of epinephrine intravenously	1.5	6.0	1.5	6.0	Total of 60 min.	Marked
	4 mg. of mecholyl intravenously	1.5	6.0	1.0	4.5		Marked

* Paracentesis: 3 minims (0.18 cm.) of aqueous removed from the anterior chamber, followed by the injection of epinephrine hydrochloride within four minutes.

TABLE 2.—*Effect of Nitrites on the Caliber of the Retinal Vessels*

Animal No.	Quantity of Medication	Caliber of Vessels in Terms of Micrometer Readings: Before Medication		After Medication		Length of Time Observed, Minutes	Systemic Reaction
		Artery	Vein	Artery	Vein		
2	40 mg. of sodium nitrite intravenously	2.5	4.5	2.5	4.5	25	Slight
6	80 mg. of sodium nitrite intravenously	2.0	4.5	2.0	4.5	25	Moderate
7	70 mg. of sodium nitrite intravenously	2.5	4.5	2.5	4.5	20	Moderate
11	40 mg. of sodium nitrite intravenously	2.0	4.5	2.0	4.5	25	Slight
33	1 ampule of amyl nitrite	3.0	5.0	3.0	5.0	25	Marked
35	1 ampule of amyl nitrite	2.0	5.0	2.0	5.0	30	Marked
34	3 minims of glyceryl trinitrate intravenously	2.5	5.0	2.5	5.0	25	None

TABLE 3.—*Effect of Mecholyl on the Caliber of the Retinal Vessels*

Animal No.	Quantity of Medication	Caliber of Vessels in Terms of Micrometer Readings: Before Medication, Artery	Before Medication, Vein	After Medication, Artery	After Medication, Vein	Length of Time Observed, Minutes	Systemic Reaction
36	Mecholyl chloride, 3 mg. intravenously	2.0	4.0	1.5	2.5	25	Marked
37	Mecholyl chloride, 3 mg. intravenously	2.0	5.0	1.5	2.5	25	Marked
39	Mecholyl chloride, 3 mg. intravenously	2.0	4.0	1.5	3.0	30	Marked
38	Mecholyl chloride, 4 mg. intravenously	1.5	4.0	1.5	4.0	30	Marked
40	Mecholyl chloride, 4 mg. intravenously	1.5	5.5	1.0	3.0	35	Marked
41	Mecholyl chloride, 4 mg. intravenously	2.0	4.0	2.0	4.0	30	Marked
42	Mecholyl chloride, 4 mg. intravenously	2.0	3.0	1.0	1.5	30	Marked
43	Mecholyl chloride, 4 mg. intravenously	2.5	4.5	1.5	3.5	25	Marked
44	Mecholyl chloride, 4 mg. intravenously	2.0	3.0	1.5	2.0	25	Marked
47	Mecholyl chloride, 4 mg. intravenously	2.0	4.0	1.5	3.0	30	Marked
45	Paracentesis*	2.0	3.5	2.0	3.5	4	
	Mecholyl chloride, 3 mg. intravenously	2.0	3.5	1.5	2.5	45	Marked
46	Paracentesis*	2.0	3.5	2.0	3.5	4	
	Mecholyl chloride, 3 mg. intravenously	2.0	3.5	1.5	2.5	45	Marked
64	Mecholyl chloride, 10 mg. subcutaneously	2.0	4.5	1.0	3.0	45	Marked
49	Paracentesis†	1.5	6.0	1.5	6.0	4	
	Epinephrie hydrochloride, 1 cc. 1:1,000	1.5	6.0	1.5	6.0	Total of 60 min.	Marked
	Mecholyl chloride, 4 mg. intravenously	1.5	6.0	1.0	4.5		Marked
16	Mecholyl chloride, 5 mg. retrobulbarly	1.5	4.0	1.5	4.0	20	Marked
48	Ethyl ester of betamethylcholine, 8 mg. intravenously	1.5	3.0	1.5	3.0	30	Moderate
50	Ethyl ester of betamethylcholine, 10 mg. intravenously	2.0	4.5	2.0	4.5	30	Moderate

* Paracentesis: 3 minims of aqueous removed from the anterior chamber, followed by the injection of mecholyl within four minutes.

† Paracentesis: the same as with animal 49 in the experiment with epinephrine hydrochloride.

TABLE 4.—*Effect of Subconjunctival Injection of Mecholyl on the Caliber of the Retinal Blood Vessels*

Animal Number	Quantity of Mecholyl Bromide Injected Subconjunctivally, Mg.	Caliber of Vessels in Terms of Micrometer Readings: Before Medication, Artery	Before Medication, Vein	After Medication, Artery	After Medication, Vein	Length of Time Observed, Minutes	Systemic Reaction
56	6	1.5	3.5	2.0	4.5	35	Moderate
59	8	2.0	4.0	2.0	4.0	40	Moderate
60	8	2.0	4.5	2.0	4.5	35	Moderate
61	8	1.5	4.0	1.5	4.0	30	Moderate
62	10	2.0	5.0	2.0	5.0	30	Marked

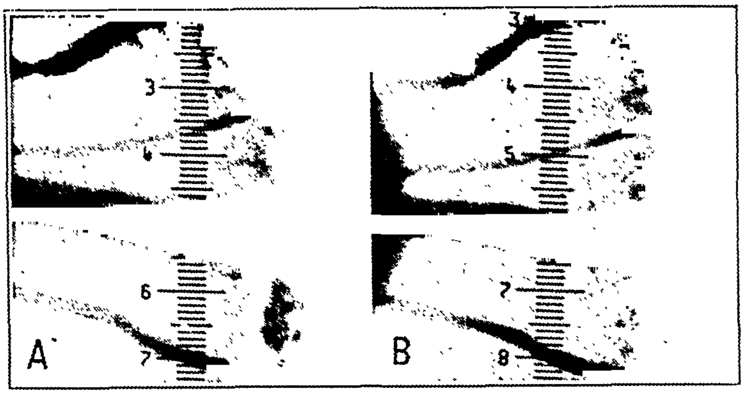

Fig. 4 (animal 37).—The effect of an intravenous injection of mecholyl on the caliber of the retinal blood vessels. *A*, before injection; *B*, after injection.

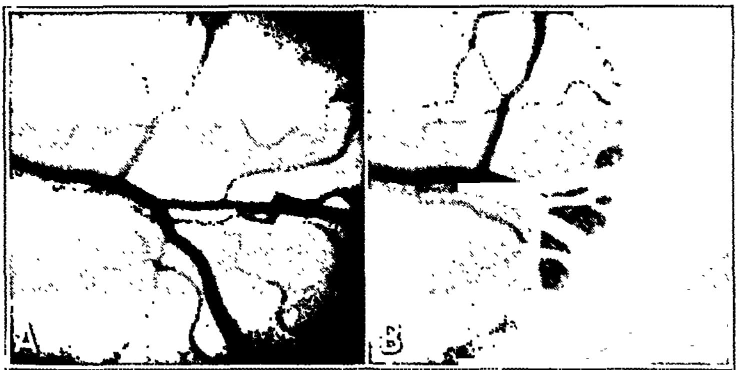

Fig. 5 (animal 56; table 4).—The effect of a subcutaneous injection of mecholyl on the caliber of the retinal blood vessels. *A*, before injection; *B*, after injection.

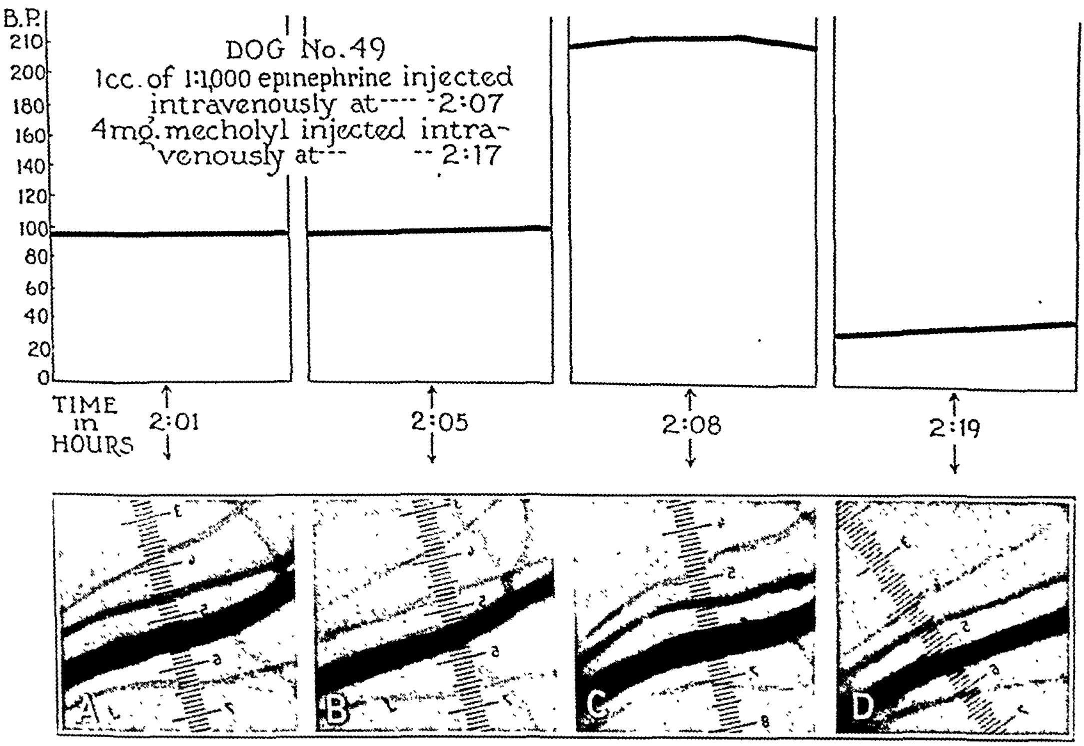

Fig. 6 (animal 49).—The effect of paracentesis together with subsequent injections of epinephrine and mecholyl. *A*, normal appearance; *B*, after paracentesis; *C*, after injection of epinephrine; *D*, after injection of mecholyl.

mecholyl bromide subconjunctivally. The evidence presented in table 3 shows a contraction of both the artery and the vein in 11 animals after injection into the femoral vein (fig. 4) and a dilatation of the artery and vein in 1 after subconjunctival administration (table 4 and fig. 5). The vasoconstriction closely paralleled the decrease in blood pressure, and this response was not altered in 3 animals in which paracentesis was performed at the beginning of the experiment (fig. 6).

The ethyl ester of betamethylcholine was injected intravenously into 1 dog and subcutaneously into another; no change was recorded in the caliber of the vessels.

COMMENT

It is important to recall that the circulation of the eye, like that of the brain, occurs within a closed space. The walls of this space, i. e., the tunic of the eye, are elastic to only a slight degree. Since marked variations in the level of the systemic blood pressure are transmitted directly to the retinal vessels, it becomes obvious that the changes in the caliber of the retinal vessels must, of necessity, depend on one or more of the following factors: (1) histologic structure and contractile elements of the vessels, (2) changes in the intraocular pressure and (3) level of the general blood pressure.

For the study of the structure of the retinal vessels, much credit is due Friedenwald,[62] Bailliart [63] and Lambert. According to Friedenwald, the central artery of the retina, posterior to the cribiform plate, has an average internal diameter of 0.1 mm. The intima and adventitia are well formed at this level, and the internal elastic lamella is present. Like similar arteries of its size, the thickness of the media is about one-fifth the diameter of the lumen, and the muscle fibers are easily discernible. The internal caliber of the vessel remains the same as it passes through the cribriform plate, but the thickness of the wall of the vessel is abruptly reduced to less than one-half its former thickness. Likewise, the intima and the internal elastic lamella are reduced to single layers and later disappear beyond the primary branches. The media are reduced to about 10 microns, and histologically it is difficult to demonstrate contractile elements. Similarly, it has been shown by Schäfer [64] that the veins of the retina resemble capillaries and consist of a single layer of endothelial cells without musculature.

There is little doubt but that the atonic veins reflect passively the fluctuations in the general blood pressure. The arteries, however, are

62. Friedenwald, J. S., in Cowdry, E. V.: Arteriosclerosis, New York, The Macmillan Company, 1933, p. 363.

63. Bailliart, P.: The Retinal Circulation, Chicago, Professional Press, Inc., 1928.

64. Schäfer, quoted by Bailliart.[63]

capable of contraction, because spasm of the central artery is frequently encountered. These spasms are possibly caused by stimulation of the contractile cells of Rouget. It is also possible that certain of these cells may be differentiated and possess the power of dilatation. All these things are without proof, but if such reactions do take place, they must necessarily occur when there is a certain degree of equilibrium established between the intraocular and the arterial tension. It is not inconsistent to believe that sharp shifts in this equilibrium could completely mask the feeble actions of such cells.

It is known that there is an intimate relation between the fluctuations of the general blood pressure and the intraocular tension. In ordinary experimental work these reactions parallel each other, but recently it has been shown by Duke-Elder [65] that there is a certain degree of dissociation between these reactions. This dissociation is demonstrated by the effect of amyl nitrite. After inhalation of this drug there is a sharp drop in systemic blood pressure, with an increase in intraocular tension, which is apparently caused by dilatation of the capillary bed.

The complexity of these reactions has also been shown by Duke-Elder following the use of epinephrine. Epinephrine in small doses (1 cc. of a 1: 10,000 solution) or in doses sufficiently large to cause a rise in blood pressure, results in an increase in intraocular tension. With large doses (1 :1,000 solution) there is a marked elevation of general blood pressure, and intraocular tension decreases. The lowering of the intraocular tension, in the face of increased arterial pressure, is explained on the basis of vasoconstriction of the capillary bed of the choroid.

Duke-Elder has likewise shown the effect of acetylcholine on the intraocular tension. With small doses this drug produces a decrease in intraocular as well as in arterial tension, but with large doses the intraocular tension rises secondarily to a contraction of the extraocular muscles. When tenotomy is performed on the rectus muscles, the ocular tension drops.

The relation of these complex reactions to this experimental work is clarified by recognizing that under normal conditions all of these variable forces exist within a closed space in a state of equilibrium. The two most important opposing forces within this equilibrium are arterial tension and intraocular pressure. Passive dilatation occurs when the arterial tension is markedly increased and the intraocular tension lags behind.

Thus, in the experiments with epinephrine the vasodilatation occurred at the crest of the rise in blood pressure. As Duke-Elder has shown, this reaction was probably facilitated in some cases by a decrease in ocular tension, but the relatively greater incidence of negative

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65. Duke-Elder, S.: Recent Advances in Ophthalmology, Philadelphia, P. Blakiston's Son & Co., 1934, p. 115.

response, even in the face of paracentesis, certainly speaks in favor of a maximal dilatation of the vessels at the time the medication was administered. It does not seem logical to suppose that the inherent contractile forces played any part in these negative responses, since the small doses of epinephrine and the retrobulbar injections brought about no constriction of the vessels. The foregoing results and considerations confirm the work of Lambert and of Riser, Couadau and Mériel.

A reversal of the forces within this equilibrium tends to produce vasoconstriction. In the case of the nitrites the negative responses were possibly caused by insufficient doses of the drug. Since tracings were not made of the blood pressure in the experiments in which the nitrites were used, it is unwarranted to adduce further conclusions.

The overwhelming effect of a marked decrease in the systemic blood pressure is impressively demonstrated in the experiments with mecholyl, in which a large dose of the drug was administred either intravenously or subcutaneously. The intensity of this reaction was not impaired by paracentesis, and the spasm of the ocular muscles was at no time sufficiently great to overcome this decrease in ocular tension. These findings unquestionably refute any assumption that large doses in conjunction with paracentesis can produce vasodilatation of the retinal vessels.

In order to demonstrate expansile elements in the vessels themselves, it is necessary to have an optimum balance existing between the two aforesaid major forces. Such a balance was present in the experiments with mecholyl in which the drug was given subconjunctivally near the site of its reaction. Vasodilatation was recorded in only 1 animal in this group (fig. 5). This response apparently proves that the retinal vessels do possess a weak inherent expansile mechanism, but it is exceedingly difficult to reconcile the histologic knowledge of the structure of the vein with its increase in caliber. As in the previous experiment, the old issue of maximal dilatation caused by the anesthetic is still present. It is known that intraperitoneal injections of pentobarbital sodium produce peripheral vasodilatation. (Richter and Oughterson [66] and Gruber and Roberts [67] found dilatation of the cerebral vessels with the use of barbiturates.) The effect of pentobarbital sodium, however, on the retinal blood vessels is unknown. In this work it was impossible to invalidate this argument because of the technical difficulties associated with photography of the unanesthetized animal.

Practical Application.—So far as animal experimentation is applicable to man, this work casts doubt on the efficacy of using large doses of vasodilators for the treatment of spasm and closure of the

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66. Richter, H. G., and Oughterson, A. W.: J. Pharmacol. & Exper. Therap. **46**:335, 1932.

67. Gruber, C. M., and Roberts, S. J.: J. Pharmacol. & Exper. Therap. **27**: 349, 1926.

central vessels of the retina. It must be borne in mind, however, that there was an increase in the caliber of the arteries and veins in 1 experiment with mecholyl in which the drug was given subconjunctivally, but the inherent vasodilator mechanism is so weak that vasodilatation is to be expected in only a small percentage of cases.

Physiologically, it is more sound to perform paracentesis and inject epinephrine subcutaneously, but few clinicians will subscribe to the use of doses large enough to produce this effect.

Thus, it is evident that the clinical aspect of this problem will be solved only when physicians realize that photographs constitute a more conclusive form of evidence than ordinary ophthalmoscopic observation. Until such proof is supplied, animal experiments of this nature should assume some degree of priority.

SUMMARY

Methods for photographing the retinal blood vessels have been discussed.

Experimental observations of various investigators have been cited which indicate the discrepancies in the literature concerning the responses of these vessels to epinephrine, nitrites and choline derivatives.

New photographic evidence has been presented with the following findings:

(*a*) Injections of epinephrine hydrochloride into 16 dogs produced a questionable dilatation of the artery in 2 animals, with an increase in the caliber of the veins in 7.

(*b*) Nitrites were administered to 7 dogs with negative results.

(*c*) Injections of mecholyl into 20 dogs produced a decrease in the caliber of the vessels in 11 and an increase in 1.

(*d*) Ethyl ester of betamethylcholine was injected into 2 dogs, with no response.

(*e*) No increase in the caliber of the vessels was found after paracentesis.

The efficacy of treating occlusion of the central arteries with vasodilators has been questioned.

A plea has been made for more photographic studies of the reactions of the retinal blood vessels in man.

The mecholyl used in this study was supplied by Merck & Co., Inc.

BIOMICROSCOPY OF CICATRICES AFTER IRIDECTOMY AND THE OPERATION OF ELLIOT OR OF HEINE

P. T. ARCHANGELSKY, M.D.
TASHKENT, U.S.S.R.

Theoretic investigations often precede practical successes. Sometimes, however, it is a long time before a thorough theoretic basis and an exhaustive explanation for practical successes are available. Such is the case when one tries to explain the results obtained with iridectomy and with the operation of Elliot or that of Heine.

Ophthalmologists are right in associating the reduction of intraocular tension with the structure of the filtration pillow and the postoperative cicatrix, because this is the most essential point for the solution of the problem.

However, a number of details in the structure of the pillow and cicatrix have escaped the attention of those investigators who tried to solve the problem by the macroscopic method. The same thing happened to those who approached this question with only the microscopic method.

The third method of investigation which is now available is macromicroscopy, e. i., biomicroscopy. This new method enables one to observe all the details which formerly escaped attention.

Biomicroscopic examination of cicatrices left after the operations of Elliot or of Heine revealed a conformity to the same established law that vessels developed in the region of the cicatrices. As to the disposition of these vessels, it is almost the same as that observed around a foreign body or an infiltrate.

My observations are rather similar to those published by Sondermann[1] in 1934, but the collection of my data began in 1931.

This collection embraces the data on patients operated on by the method of Elliot or of Heine. Some of the patients were subjected to iridectomy.

Notwithstanding the different methods of surgical treatment, the biomicroscopic pictures of the disposition of the blood vessels in the region of postoperative cicatrices were essentially the same.

From the ophthalmologic clinic of the Molotov's Tashkent Medical Institute, Dr. P. F. Archangelsky, director.

1. Sondermann, E.: Meine Glaukomtheorie und die Klinik des Glaukoms, Klin. Monatsbl. f. Augenh. **92**:313 (March) 1934.

This fact gives ground to the statement that in these basic methods of surgical treatment of glaucoma the mechanism of the reduction of the intraocular tension is uniform.

The biomicroscopic pictures observed follow:

CASE 1.—P., a woman aged 63, was subjected to iridectomy because of an acute attack of glaucoma in October 1934. The examination made on July 7, 1938, showed that a pillow had developed in the region of the postoperative cicatrix; blood vessels, coming from the conjunctival, the episcleral and the pericorneal network, approached this pillow and surrounded and penetrated it. The vascularity of the pillow was especially pronounced when massage was applied to the region of the postoperative cicatrix. (fig. 1).

CASE 2.—G., a man aged 49, in 1934 was operated on by the method of Elliot because of chronic glaucoma. Biomicroscopic examination in 1938 showed a

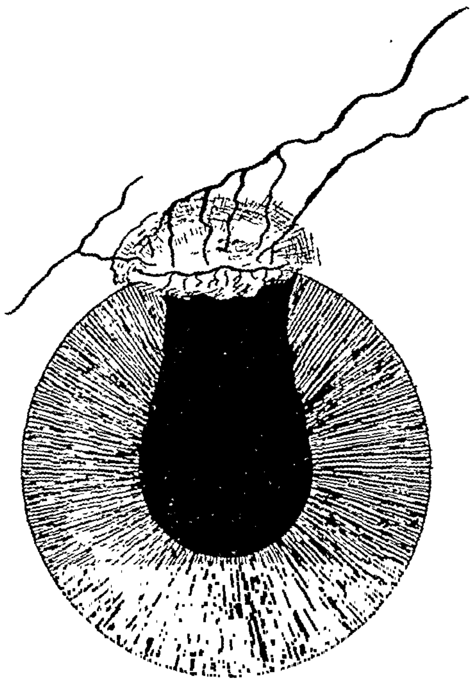

Fig. 1 (case 1).—Biomicroscopic picture of a cicatrix occurring after iridectomy.

well developed pillow pierced by blood vessels; one of these vessels produced an ampulla-like dilatation. Under low magnification the movement of the blood in this pillow reminded one of the movements of pink worms in a clod of moist soil; under high magnification the movement of erythrocytes was similar to the movements of ants in an anthill (fig. 2).

CASE 3.—N., a woman aged 40, was operated on by the method of Heine because of absolute glaucoma. Two months later biomicroscopic examination revealed that vessels with brushlike endings approached the postoperative cicatrix and surrounded it. In the angle of the anterior chamber could be seen a vessel, which left the chamber after making a loop on entering it (fig. 3).

I cite here only 3 cases, but my investigations by the biomicroscopic method embrace 50 cases in which the operation of Elliot was done, 20

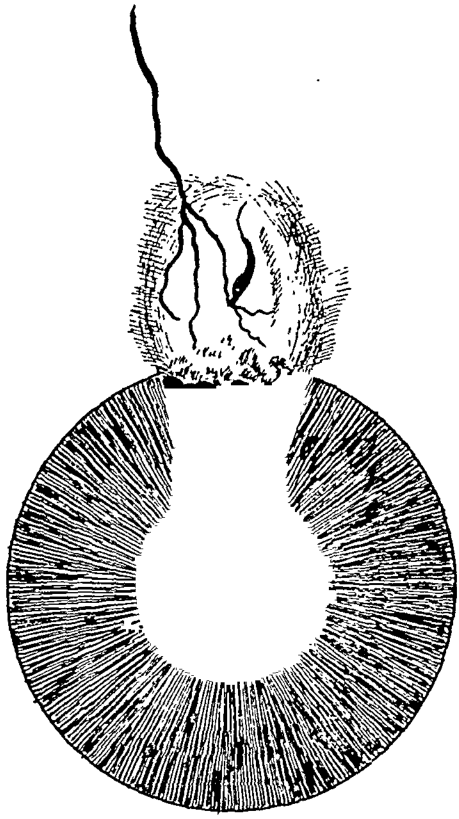

Fig. 2 (case 2).—Biomicroscopic picture of a cicatrix occurring after the operation of Elliot.

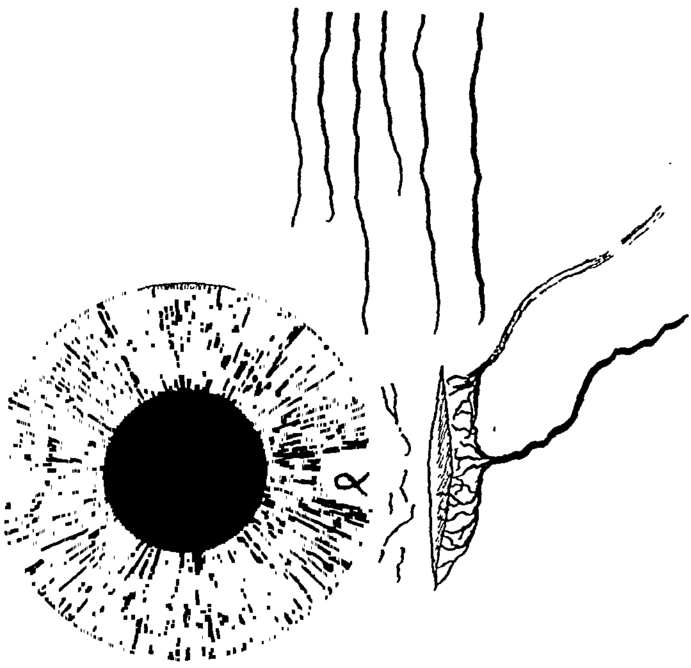

Fig. 3 (case 3).—Biomicroscopic picture of a cicatrix occurring after the operation of Heine.

in which the operation of Heine was performed and some cases in which iridectomy was employed. My observations lead to the following conclusions:

1. The disposition of the vessels developing in the region of the cicatrix left after iridectomy and after the operation of Elliot or of Heine conforms to some established law.

2. These vessels form the additional system for the outlet of intra-ocular fluid.

ALCOHOL-TOBACCO (TOXIC) AMBLYOPIA TREATED WITH THIAMIN CHLORIDE

LORAND V. JOHNSON, M.D.
CLEVELAND

Toxic amblyopia, with centrocecal scotomas and relatively larger scotomas for red and green, and commonly with peripheral neuritis and hypochlorhydria, appears to be generally accepted to be associated with dietary deficiencies of the vitamin B complex. The more rapid clinical improvement following the use of brewers' yeast or other forms of vitamin B [1] appears generally to be sufficiently satisfactory to cause it to be considered a routine therapeutic agent.

Admitting the multiple deficiencies from which these patients suffer, I desired to ascertain which of the vitamin B factors, if any, singly would give results most approaching the clinical improvement following the use of brewers' yeast. Thiamin chloride (B_1) and nicotinic acid appeared to be the most probable factors in the B complex and were therefore used.

CASE 1.—The first patient seen had centrocecal scotomas for red and green in each eye, a small scotoma in the left eye for white (1 mm. test object at 1,000 mm.) and vision of 20/70 in the right eye and 20/100 in the left eye. He was advised to abstain from alcohol and tobacco and to take 12 mg. of thiamin chloride daily. He cooperated well, and after five weeks the vision was 20/30.

CASE 2.—The patient had a large centrocecal scotoma (nearly 30 degrees) and considerable reduction in vision for red and green. He had been treated several times for acute alcoholism. Suggestive atrophy of the papillomacular bundle of the optic nerve was present. He was admitted to the University Hospital on Oct. 10, 1937, and was given a diet high in B vitamins and in addition 60 mg. of nicotinic acid daily. On October 24 his vision had not improved, but there was definite atrophy of the papillomacular bundle. Thiamin chloride, 12 mg. daily, was given instead of the nicotinic acid, but no improvement in vision followed. When the patient was last seen there was almost complete atrophy of the optic nerve.

CASE 3.—A gardener had been dismissed a month previously because of incompetence, it being assumed that his poor work was due to his intoxication while working. His consumption of liquor then increased in proportion to his leisure time. When he was first seen his scotomas were about 15 degrees for a

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From the Department of Surgery, Ophthalmological Service, of the Western Reserve University School of Medicine and the University Hospitals.

1. Carroll, F. D.: Importance of Diet in the Etiology and Treatment of Tobacco-Alcohol Amblyopia, Arch. Ophth. **18**:948 (Dec.) 1937.

5 mm. test object at 1,000 mm. Six weeks of abstinence plus the use of 60 mg. of nicotinic acid daily and 12 mg. of thiamin chloride daily allowed the return of vision to 20/70 and 20/100. As my supply of nicotinic acid was exhausted, the patient was advised to continue the thiamin chloride and to return to the clinic in two weeks. Instead, he returned in five weeks, stating that he had started drinking and smoking a pipe again but had continued to take the thiamin chloride until five days previously. His vision was 20/40 in each eye. He returns every month or so for additional medication. He sometimes drinks to excess. His vision remains 20/20 but with a narrow scotoma for red and green from the blindspot almost to the macula. He has been followed for six months since the recovery of vision to 20/40 and has not since reported any symptoms of peripheral neuritis.

CASE 4.—A professional man, unmarried and without relatives, stated that he had been drinking excessively only since the death of his mother, about six months previously. He was an inveterate cigar smoker. His scotoma for white (a 5 mm. test object at 1,000 mm.) was about 8 degrees in the vertical dimension. He refused to cease smoking and drinking (largely wine), and threats of permanently impaired vision brought only replies concerning suicidal intentions. He had ceased making appointments in his office and has since closed it, and he is determined to expend his savings. He was given 100 thiamin chloride tablets and advised to take 12 mg. daily (2 tablets three times a day). In seven and a half weeks his vision was 20/40 and in nine weeks 20/30. He was determined to continue to smoke twelve to fifteen cigars daily and to drink as much wine and more whisky than before he closed his office. His vision in fifteen weeks was 20/20 with a centrocecal scotoma for red and green. He said he had never had polyneuritis.

CASE 5.—The patient received only thiamin chloride, 12 mg. daily. He ceased smoking and drinking promptly, and in seven weeks vision improved from 20/100, and 20/200 to 20/30 in each eye.

This small series of cases suggests the value of thiamin chloride in the treatment of toxic amblyopia. It does not include sufficient studies of the value of nicotinic acid for the treatment of a condition that accompanies a multiple deficiency.

INDUCED SIZE EFFECT

II. AN EXPERIMENTAL STUDY OF THE PHENOMENON WITH RESTRICTED FUSION STIMULI

KENNETH N. OGLE, Ph.D.

HANOVER, N. H.

In the first paper of this series [1] the general aspects of a new phenomenon in binocular vision, designated as the induced size effect, was discussed. The nature of this phenomenon will be clear from the following explanation:

When a change in the relative sizes of the images of the two eyes is introduced in the horizontal meridian by placing a meridional size lens before one eye, all objects in a binocular visual field relatively free from strong perspective clues appear as though rotated a certain amount from their original positions about a vertical axis through the fixation point. This apparent rotation of the binocular visual field is to be expected, for obviously it depends on the binocular depth perception that arises from horizontally disparate retinal images. Any change in the relative size in the horizontal meridian will necessarily change the disparities of the retinal images, and, accordingly, new stereoscopic depth values relative to the fixation point must arise for those images. The introduction before one eye of a meridional size lens which magnifies in the horizontal meridian only changes geometrically the disparities of the retinal images, with the result that an apparent rotation occurs for all objects seen binocularly. The direction of this rotation is such that objects on the side of the magnified image will appear farther away; those on the other side will appear nearer. This phenomenon, occurring with a difference in the sizes of the images in the horizontal meridian, is described as a geometric effect, and the magnitude of the apparent rotation as computed from geometric considerations [2] is in agreement with experiment. Thus, the magnitude of the apparent rotation increases almost proportionally to the introduced difference in the sizes of the images.

From the Division of Research in Physiological Optics, the Dartmouth Eye Institute, Dartmouth Medical School.

1. Ogle, K. N.: Induced Size Effect: I. A New Phenomenon in Binocular Space Perception Associated with the Relative Sizes of the Images of the Two Eyes, Arch. Ophth. **20**:604 (Oct.) 1938.

2. For example, see Ogle, K. N.: Die mathematische Analyse des Längshoropters, Arch. f. d. ges. Physiol. **239**:748, 1938.

Now, contrary to expectations, when a meridional size lens is placed before one eye so as to change the size of the retinal image in the vertical meridian only, objects which are seen binocularly also appear rotated a certain amount from their original positions about a vertical axis. Here, however, the rotation is in the direction opposite to that of the geometric effect; that is, objects on the side of the magnified image appear nearer; those on the other side appear farther away. It is not easy to account for this particular false interpretation of the position of objects in the binocular visual field, since no depth discrimination is known to arise from vertical disparities of the retinal images.[3] So far as the subjective binocular orientation of objects is concerned, the effect is as though the increased size of the image of one eye in the vertical meridian induced an increase in the size of the image of the other eye in the horizontal meridian. Because of the analogy, this phenomenon has been designated the induced size effect.

The two effects differ from each other in one important respect: When the image of one of the eyes is increased in the vertical meridian, the magnitude of the apparent rotation of a test plane increases with the difference in the sizes of the images only within a limited range. Reaching a maximum at these limits, the apparent rotation then actually decreases with a further increase of the difference in the sizes of the images in the vertical meridian. The graphic representation of the induced size effect is a typical elongated S-shaped curve, while that of the geometric effect, when the apparent rotation is virtually proportional to the difference in the sizes of the images in the horizontal meridian, is a straight line in the diagonally opposite quadrants of the graph. These findings definitely point to a physiologic origin of the induced size effect. Certainly, it cannot be explained solely in terms of the properties of the meridional size lenses, as is the case in the geometric effect. Hence, the induced size effect is basically different from the geometric effect.

As described in the previous paper,[1] the data showing these effects were obtained with an apparatus in which the apparent rotation of the binocular field was measured by means of a test plane which the observer could rotate about a vertical axis. The fusion pattern on the plane, as presented to the observer, consisted of a large number of ink dots scattered irregularly over both sides of a sheet of plate glass. Such a complex pattern, while adequate and valuable for demonstrating the

3. For a brief discussion and for references pertaining to the stereoscopic depth localization of vertical disparities, the reader is referred to Tschermak, A.: Optischer Raumsinn, in Bethe, A.; von Bergmann, G.; Embden, G., and Ellinger, A.: Handbuch der normalen und pathologischen Physiologie, Berlin, Julius Springer, 1930, vol. 12, pt. 2, p. 928.

phenomena, is unsuited for a more detailed and fundamental study of the induced size effect. To obtain a finer analysis, simpler and restricted fusion patterns were used on the test plane. The results of the experiments with these patterns are considered in this paper.

EXPERIMENTAL PROCEDURE

The apparatus used and the procedure followed here were essentially those of the preliminary experiments described in the first paper.[1] The difference lay only in the type of the fusion pattern on the test plane presented to the observer.

An observer, whose head is held by a suitable bite or chin cup and adjustable forehead rests, fixates the center of an object plane for a given distance in symmetric convergence (fig. 1). The object plane is free to rotate about a vertical axis through its center. The apparatus is set up so that the visual plane of the observer's eyes will be horizontal. A conveniently placed protractor, calibrated in half degrees, and an indicator permit a measurement of the rotation of the plane from the "frontal" position to an estimated tenth of a degree. The object plane usually consists of a sheet of plate glass 30 by 30 by 0.3 cm., supported in a frame. Suitable figures or contours of different types are drawn in india ink or black paint on the surface of the object plane. A large sheet of white cardboard, uniformly illuminated, suspended for a background, silhouettes the figures seen on the object plane by the observer. A shield of white cardboard mounted at a distance of about 10 cm. before the eyes restricts the field of vision to the object plane. This shield is usually illuminated to the same brightness as the background. The observer can control the rotational orientation of the object plane by means of a cord.

To measure the apparent rotation of the binocular visual field, which is brought about by a meridional size lens before one eye, the observer rotates the test plane until it appears in the same position in which it had been before the difference in the sizes of the images was introduced. This rotation is obviously in the direction opposite to that of the apparent rotation of a fixed plane. In all the experiments to be described here the observer adjusts the object plane so that it appears normal to the direction of its center from him; that is, he adjusts it for an apparent "frontal" position. During a given experiment a number of adjustments of the plane are made for each different combination of meridional size lenses used before the eyes.

In simplifying the fusion pattern, several considerations must be taken into account. The main objective, of course, was to obtain the simplest pattern with which the effect would occur and yet one in which the fusion stimuli would be confined to definite areas on the test plane, hence, to specific regions on the retina. If the test plane is left entirely clear, except for a horizontal row of very small dots, no induced size effect can be found. With an increase of the size of the dots, however, even to the extent of their subtending a visual angle of 0.5 degree, or if the row of small dots is not precisely horizontal, a small induced size effect can be demonstrated. It is clear, therefore, that the effect cannot occur unless the pattern contains vertically separated contours.

The preliminary data were taken with a pattern consisting of a large number of ink dots scattered irregularly on both sides of the entire plane. Such a pattern provides nearly a maximum of vertically separated contours. Decreasing the total number of dots does not essentially change the nature of this pattern. The scattered dots may, however, be grouped in an approximate ring with the center at the fixation point, so that the fusion stimuli are confined to a fairly definite visual angle. But such a pattern has contours extending continuously from the center to the vertical diameter of the ring, and, therefore, it could not be used as a simplified pattern for the basic investigation of the induced size effect. The disadvantages of a ring could be avoided by restricting the scattered dots to two horizontal bands above and below the point of fixation. While such a pattern does confine the fusion contours to fairly specific vertical regions, it introduces an objectionable perspective factor, which may have an influence on the apparent position of the plane.

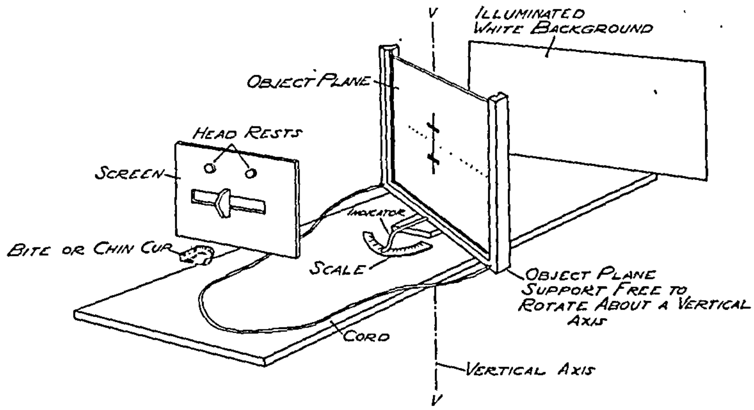

Fig. 1.—Schematic drawing of the apparatus used for a study of the induced size effect.

On the basis of these considerations it was found that the induced size effect would occur even with so simple a fusion pattern as single contours either above or below the center of the plane or both. For the various experiments described here, essentially three different patterns were used (fig. 2). In each case the row of small ink dots was used to provide the necessary horizontal stereoscopic stimuli by which the orientation of the plane about the vertical axis could be ascertained. The dots were 0.5 and 0.75 mm. in diameter and were spaced regularly,[4] about 1 cm. apart, in a horizontal row across the center of the plane. A vertical line (or thread) through the central dots assured fusion. Specifically, the patterns were as follows:

(*a*) At a given vertical distance above and below the horizontal row of dots, small black paper rectangles were fastened on the glass plane,

4. Probably an irregular spacing of these dots would have been better, for any possible empiric factor involved in the regularity would have been avoided.

symmetric with the vertical line.[5] These rectangles provide fusion contours that are simple and can be adjusted for any separation.

(*b*) A small white thread, with narrow ink rings at intervals of approximately 1 cm., fastened horizontally to the frame, was substituted for the row of dots on the sheet of glass. A black thread was again fastened vertically over the axis of rotation of the frame. Above and below the horizontal thread, identical narrow strips of heavy black cardboard were supported by a suitable framework. The inner ends of the strips, placed at equal distances from the horizontal thread, provided the desired fusion contours. The outer ends of the strips and the supports lay outside the field of vision. This pattern restricts the peripheral fusion stimuli in the vertical meridian to two definite contours, which do not rotate with the horizontal row of dots.

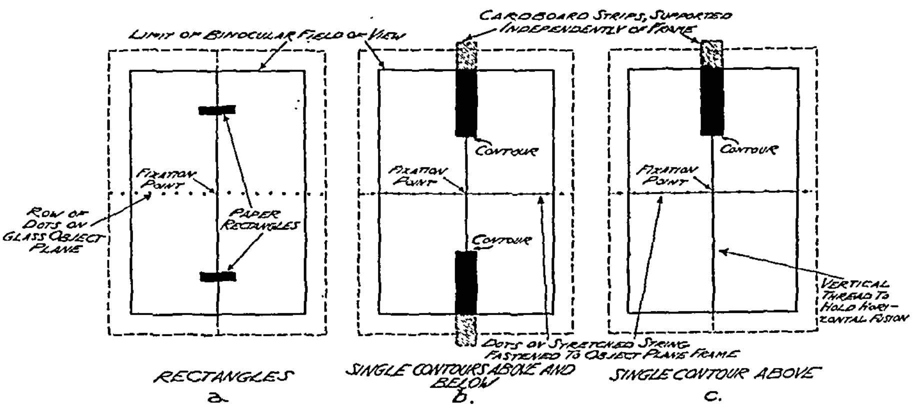

Fig. 2.—Three types of fusion patterns used in a study of the induced size effect.

(*c*) The third pattern is essentially the same as type *b*, though only one of the vertical strips is used. This limits the peripheral fusion stimuli in the vertical meridian to a single contour on one side of the fixation point.

In each of the foregoing patterns the separation of the fusion stimuli, which is necessary if the effect is to occur, is provided by the distance between the horizontal row of dots and the contours.

RESULTS

General Induced Size Effect.—It is possible with the simplified pattern to ascertain the nature of the induced size effect with greater consistency. While the experimental technic and the previous training, and perhaps even the physical condition of the observer, have an effect on the absolute magnitudes of the apparent rotations, the relative behavior

5. Small black hemispheres were substituted in some cases for the rectangles These have the advantage of not changing shape with the rotation of the plane.

of the curves is fundamentally the same for any observer. Inexperienced observers, however, may not always respond at once to so simple a fusion pattern. They may get satisfactory results only after experience has been obtained in taking data with the more complex pattern. Trained observers, on the other hand, are generally only a little less sensitive with the simple than with the more complex pattern.

The data for two observers, illustrated graphically in figures 3 and 4, were obtained by using type *b* pattern, in which the two single contours were at an angular distance of 5 degrees from the center. Table 1 gives the actual data for figure 3. Differences in the sizes of the images in the vertical meridian up to 20 per cent were introduced by accurately ground meridional size lenses [6] placed before either eye. The graphs again show all the general characteristics of the induced size effect. The apparent rotation of the test plane is, within a certain limited range, nearly proportional to the difference in the sizes of the images in vertical meridian. The slope of the line which best fits this portion of the data is defined as the maximum sensitivity of the phenomenon, being stated in degrees of rotation caused by a 1 per cent difference in the sizes of the images in the vertical meridian. Beyond the linear part of the curve, the sensitivity decreases until it becomes zero at the maximum apparent rotation. This point indicates the maximum induced size effect, and a further increase of the difference in the sizes of the images results in a decrease of the apparent rotation. These particular curves are of interest, since they show particularly how the apparent rotation decreases slowly beyond the maxima with larger difference in the sizes of images in the vertical meridian.

Of significance are the relatively small mean deviations of the mean setting of the plane in any given case. A study of table 1 shows that the average mean deviation for the given set of data is about ± 0.8 degrees, with only a few deviations exceeding 1 degree. Since the sensitivity of the apparent rotation of the plane in the geometric effect (difference in the sizes of the images in the horizontal meridian) is roughly 3.5 degrees for 1 per cent for the visual distance of 40 cm., these deviations correspond to relative variations in size of from 0.2 to 0.3 per cent. This agrees with the sensitivity of the eyes to differences in the sizes [7] of the images and again shows strikingly the extreme accuracy to which these depth perception phenomena can be measured.

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6. The meridional lenses used were especially ground so as to have no verging power for the distance of 40 cm. and no magnification in the meridian at right angles to the principal magnification meridian. For theory of design see Ogle, K. N.: The Correction of Aniseikonia with Ophthalmic Lenses, J. Optic. Soc. America **26**:323, 1936.

7. Ames, A., Jr., and Ogle, K. N.: Size and Shape of Ocular Images: III. Visual Sensitivity to Differences in the Relative Size of the Ocular Images of the Two Eyes, Arch. Ophth. **7**:904 (June) 1932.

From an extensive study of the problem it has been ascertained by trial and error that a set of data for induced size effect can be satisfactorily described algebraically (with respect to the center of symmetry) by $y = ax/(b + x^2)$, in which y is the rotation of the plane caused by a difference in the sizes of the images of x (positive for an increase in the image in the right eye) and a and b are constants. The ratio a/b specifies the maximum sensitivity. If $\bar{x}$ is the difference in the sizes of the images, expressed in percentage, for the maximum rotation $\bar{y}$, $b = \bar{x}^2$, from whence it follows that $a = 2\bar{x}\bar{y}$. For small values, the rotation

TABLE 1.—*Typical Data of the Induced Size Effect* *

Lenses Before Right Eye, Axis Horizontal		Lenses Before Left Eye, Axis Horizontal	
Difference in Sizes of Images in Vertical Meridian, Percentage L < R	Rotation of Plane, Degrees	Difference in Sizes of Images in Vertical Meridian, Percentage R < L	Rotation of Plane, Degrees
Normal	—3.7 (0.4)		
1.0	—1.5 (0.7)	1.0	— 6.0 (0.7)
2.0	+0.5 (0.7)	2.0	— 8.2 (0.2)
4.0	+2.4 (0.7)	4.0	—12.1 (0.8)
6.0	+0.8 (0.8)	6.0	—13.1 (0.8)
8.0	+0.5 (1.2)	8.0	—11.9 (0.8)
10.0	—0.7 (1.0)	10.0	—12.6 (0.7)
12.0	—0.5 (0.5)	12.0	—12.6 (0.5)
14.6	—2.1 (0.7)	14.6	—11.9 (0.4)
16.7	—2.2 (0.9)	16.7	—10.9 (0.9)
19.0	—3.7 (1.1)	19.0	—10.1 (0.6)
21.3	—3.0 (0.8)	21.3	—10.5 (1.0)

* The data for this observer (K. N. O.) were obtained with an object plane pattern consisting of two separate contours.

The data are the degrees of rotation of the plane from the true frontal position (with mean deviations) about a vertical axis (counterclockwise rotations are taken positive) when the plane is adjusted for an apparent "frontal" position for a given difference in the sizes of the images in the vertical meridian introduced by meridional size lenses. The visual distance equaled 40 cm.

These data are the mean of two individual sets of data. The mean deviations given are the average of the mean deviations of each set.

The slope of the straight line portion of the data, that is, the maximum sensitivity equaled 2.7 degrees for a 1 per cent difference.

The average difference in the sizes of the images in the vertical meridian for maximum rotation equaled 5.5 per cent.

The average maximum rotation of the test plane was 7.5 degrees.

The center of symmetry of data equaled —6.3 degrees and —1.2 per cent.

of the plane is directly proportional to the difference in the sizes of the images, while for larger values the denominator of the fraction becomes more and more dominant and the rotation decreases asymptomatically with a horizontal axis. The curves drawn to represent the data in figures 3 and 4 (and subsequent data) have been computed from this equation, suitable values for the constants a and b being used. It must be borne in mind that this relation is used *only* as a device to provide uniformity in the representative curves, and it might not be applicable to data obtained with differences in the sizes of the images greater than those used in these experiments. Moreover, the equation is used here without regard to any theoretic implications.

A study of the data in figures 3 and 4 is of special interest from the point of view of vertical disparity of the retinal images instead of from the point of view of differences in the sizes of the images in the vertical meridian.

Since in these experiments the contours for stimuli to fusion in the vertical meridian are confined to a specific visual angle, the result of increasing the size of the image in one eye in the vertical meridian is to

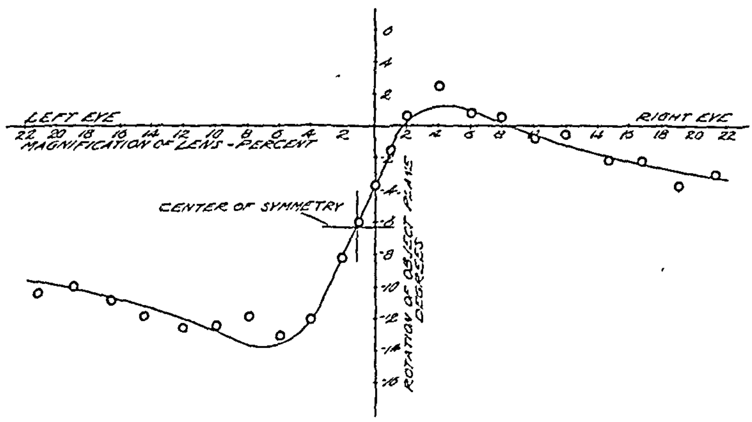

Fig. 3.—A graphic illustration of a typical set of data (for K. N. O.) showing the induced size effect for large differences in the sizes of images when the fusion in the vertical meridian is restricted to two separate contours. The visual distance equaled 40 cm.

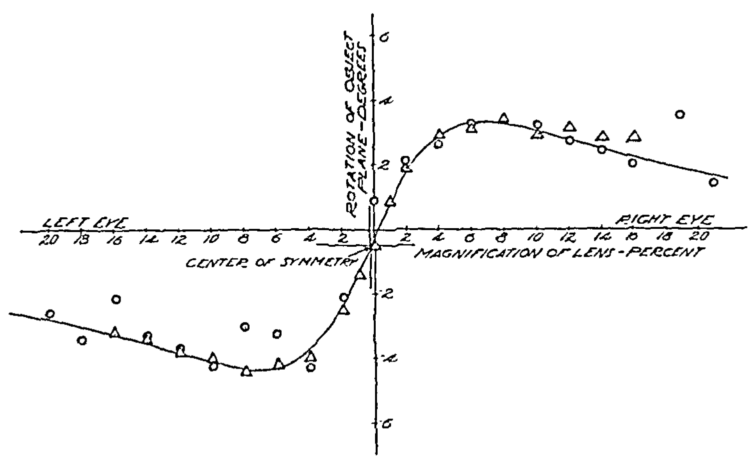

Fig. 4.—A graphic illustration of a typical set of data (for G. S. N.) showing the induced size effect for large differences in the sizes of the images when the fusion in the vertical meridian is restricted to two separated contours. The visual distance equaled 40 cm.

introduce a vertical disparity between the retinal images of the contours. This situation is illustrated schematically in figure 5, in which the eyes are shown fixating the horizontal row of dots, while the contour above (and likewise the one below) provides a peripheral stimulus to fusion in the vertical meridian. Without a size lens before the left eye, the retinal images of these contours would under normal conditions fall on ver-

tically corresponding retinal elements. The introduction of the size lens before the left eye, however, displaces the retinal image of the contour in that eye downward, so that the contour to that eye appears higher than the corresponding contour in the right eye. This difference in position of the retinal images of the same contour constitutes a vertical disparity of the retinal images, which is usually expressed by a small visual angle (δ in fig. 5). The magnitude of the disparity will be directly proportional to the magnification of the size lens and to the distance of the contour from the horizontal row of dots.

For small changes in the sizes of the images, the apparent rotation of the test plane is nearly proportional to the disparity of the retinal images. As the magnitude of the disparity increases, the increase in the apparent rotation becomes smaller, until it finally stops. Obviously, the observer is still responding to an increasing disparity of the retinal

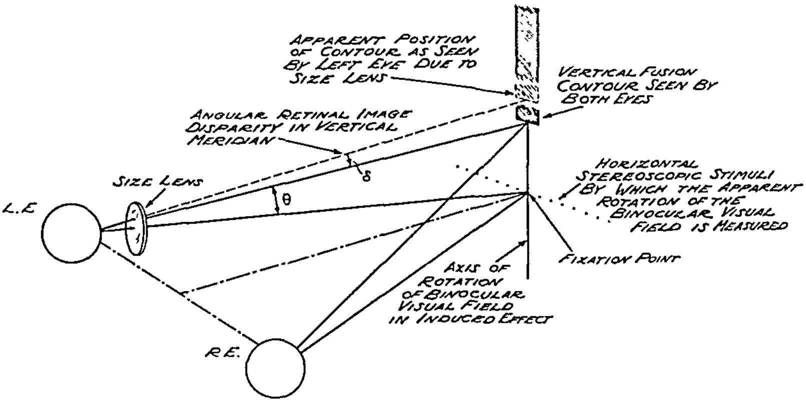

Fig. 5.—Schematic diagram showing the equivalent angular disparity of the retinal images caused by the difference in the sizes of the images introduced by a size lens.

images, beyond the point of maximum apparent rotation, though that response is decreasing slowly.

It would be of importance to know the disparity at which the response ceases entirely. Since this is difficult to ascertain experimentally, only inferences can be drawn from the data at hand. On the assumption that the analytic expression represents the data, extrapolation would indicate that although the response decreases, it never ends entirely. If an extrapolation is made for the difference in the sizes of the images at which the magnitude of the induced size effect is of the order of the precision of measurements of the experiments, the disparity is about twice the distance of the contours from the row of dots. While this estimate is probably much too high, even the magnitude of the disparity indicated at the ends of the experimental curves transcends any known retinal physiologic unit or area, in the sense of a fusion area. Such magnitudes would tend to imply, therefore, that the induced effect is the result of certain cortical processes.

One might suspect that the behavior of the induced curve beyond the maximum rotations is a fatigue phenomenon. However, the data of several experiments showed the same characteristic curve, regardless of the order in which the data were taken or the length of time employed for each setting.

A similarity can be found between the decrease of the induced size effect with large differences in the sizes of the images in the vertical meridian (or disparities of the retinal images) and the results of certain experiments with fusional movements.[8] As is known, vertical fusional movements, in the sense of a vertical divergence, can be caused by a vertical displacement of the stimulating images. Up to a certain point these movements are proportional to the initial displacement of the images. Beyond this point the resulting ocular movements lag farther and farther behind the displacement of the stimulating images, indicating that the fusion stimulus exerted by the images decreases with the amount of this displacement. In the induced size effect, a similar weakening of the fusion strength for large disparities of the retinal images can be assumed. Such a weakening might account for the S-shaped curve so characteristic of the induced effect, if that effect results from a fusion effort. Within certain limits the fusion impulse is strong enough to cause a full effect, while beyond these limits the weakening of the fusion stimuli result in a steady decrease of the effect. Under given experimental conditions, then, the influence of the vertical disparity of the retinal images introduced by the size lenses would never cease entirely, though the magnitude of the apparent rotation of the binocular visual field is eventually below the precision of measurement.

Influence of Changing the Vertical Separations of the Fusion Contours.—The results described show that the induced size effect occurs with a simple fusion pattern consisting of as little as a single contour separated from the horizontal row of dots; thus, one must infer that the effect is a response to a simple disparity of retinal images confined to relatively small areas on the two retinas. The question then arises: How does the effect vary for different distances of the contours from the fixation point? That is, how does the effect vary as a response to disparities of the retinal images at different excentric parts of the retinas?

For this study, the separated rectangles (*a*) and the single contour (*c*) types of patterns were used at a visual distance of 40 cm. In the first case a series of test planes were prepared in which the rectangles were spaced so as to subtend visual angles of 1 degree and 2.1, 3.5, 5, 7.2, 9.2 and 11.4 degrees both above and below the horizontal row of

8. Burian, H.: Fusional Movements: Role of Peripheral Retinal Stimuli, Arch. Ophth. **21**:486 (March) 1939.

dots. With rectangles 2 cm. by 0.2 cm., spaced 3.5 cm. from the horizontal row of dots for a visual angle of 5 degrees (visual distance equals 40 cm.), as the standard, the size of the small rectangles for the other planes was increased in proportion to the visual angle. In the second case the series of suspended strips of black paper provided single fusion contours for visual angles of 1 degree and 2, 3.5, 7, 9 and 12 degrees from the horizontal row of dots. The width of the strips in every case was 8 mm.

Data for S type curves for each of these patterns were obtained by three observers.[9] Differences in the sizes of the images in the vertical meridian up to 10 and 12 per cent were used. The data illustrating the results of the two patterns for two observers are shown in figures 6 and 7. The symmetric curve in each figure best fits the experimental points as a whole and is consistent with the analytic expression given previously. From each of the curves the following significant characteristics are ascertained, either by inspection or by determination from the constants of the analytic curves:[10] (1) the maximum sensitivity, given by the slope of the line which best fits the central portion of the curve; (2) the difference in the sizes of the images in the vertical meridian for which the maximum rotation[11] has occurred; (3) the maximum rotation[11] of the object plane; (4) the center of symmetry of the curve, specified by a percentage of difference in the sizes of the images, and a rotational position of the object plane.

The derived values for all the observers are given in table 2.

Maximum Sensitivity: The values for the maximum sensitivity (degrees of rotation for a 1 per cent difference in the sizes of the images in the vertical meridian at the center of symmetry) for the various patterns subtending different visual angles to the fusion contours are given for the various observers in the first row of the table. They

9. The ocular characteristics of the three observers follow: for K. N. O., vision of 20/15 in the right eye and 20/15 in the left eye (binocularly accepted a + 0.25 D.), with 3 degrees of esophoria for near vision; for R. H. D., vision of 20/15 in the right eye with a + 0.25 sph. ⊃ + 0.25 cyl., axis 90 and 20/15 in the left eye with a + 0.50 sph.; also 2.25 degrees of exophoria for near vision; for G. S. N., vision of 20/15 in the right eye with a + 0.75 sph. and 20/15 in the left eye with a + 0.87 sph.; also orthophoria (± 0.5 degrees) for near vision. The data for all observers were taken without refractive correction.

10. It is believed that a more consistent series of derived values for these characteristics can be obtained by the method used here, viz., that of finding the constants of the analytic curve which fits each set of data as a whole rather than any other method of inspection.

11. Because of the fact that the center of symmetry of the induced size effect curve for many observers is displaced from the origin of the graph, the expressions "difference in the sizes of the images for the maximum rotation of the test plane" and "the maximum apparent rotation of the test plane" will refer to the average values or to those measured from the center of symmetry.

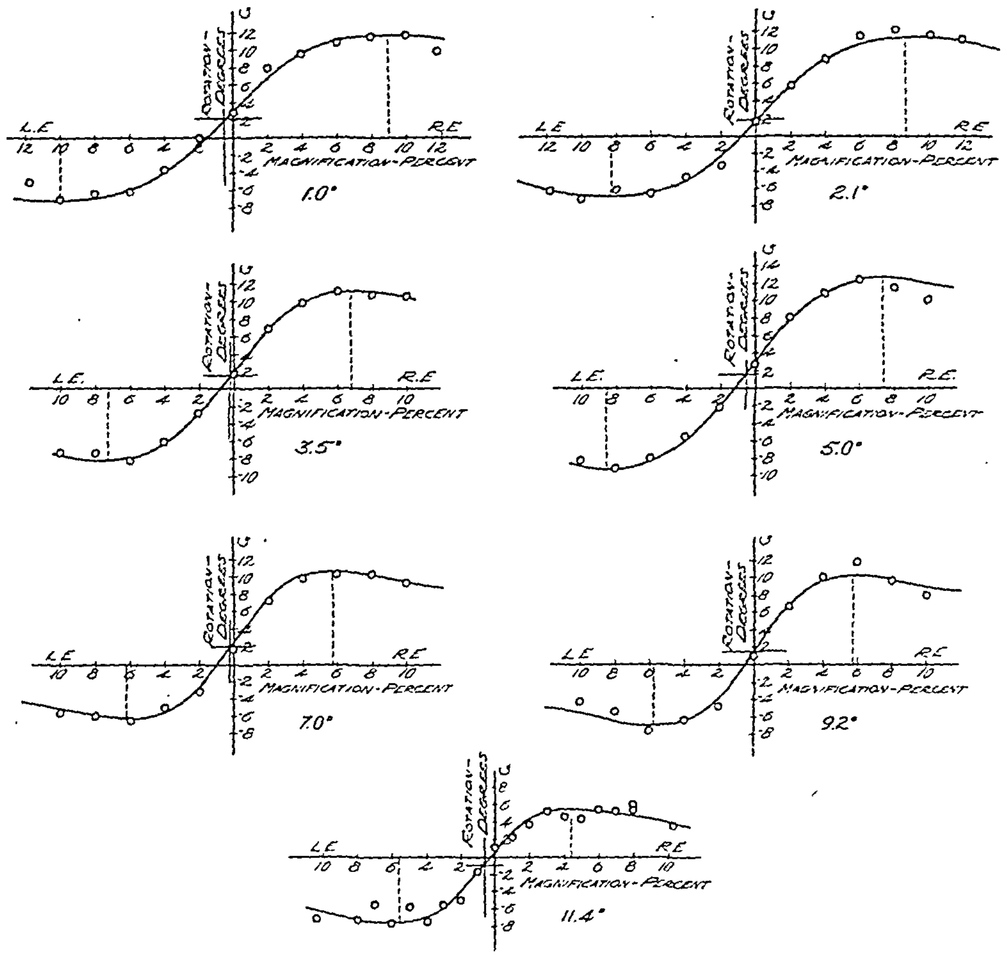

Fig. 6.—Graphic illustration of data (for R. H. D.) showing the induced size effect for different vertical angular distances of the fusion contours from the fixation point. The data are the apparent rotation (degrees) of the object plane caused by differences in the sizes of the images in the vertical meridian (percentage) introduced by meridional size lenses. The visual distance equaled 40 cm., and the pattern consisted of two rectangles.

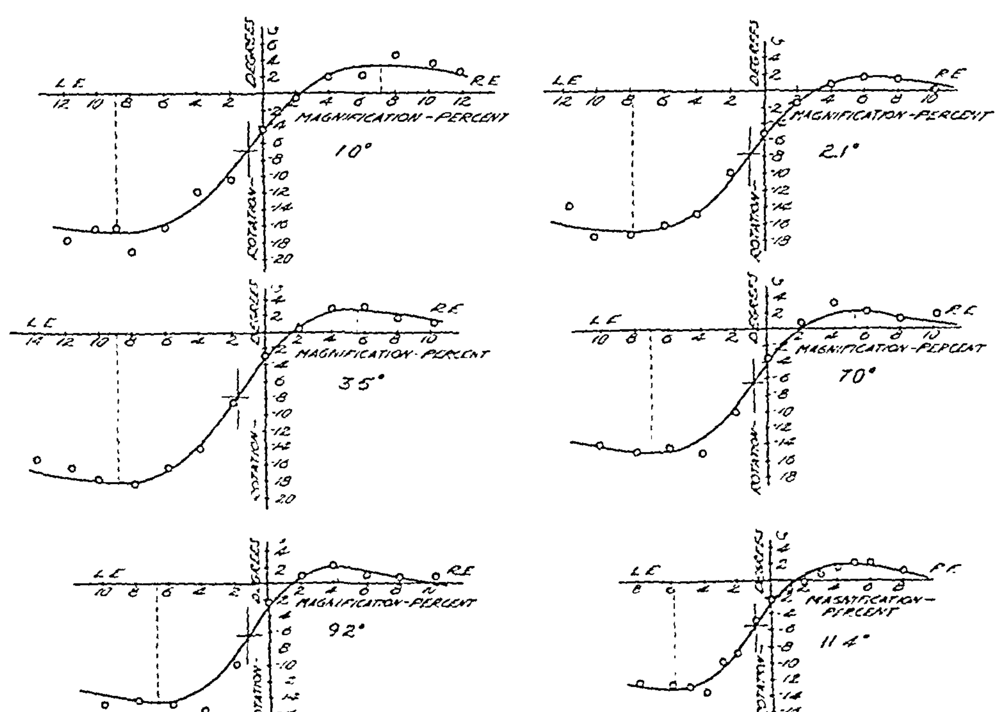

Fig. 7.—Graphic illustration of data (for K. N. O.) showing the induced size effect for different vertical angular distances of the fusion contours from the fixation point. The data are the apparent rotations (degrees) of the object plane caused by differences in the sizes of the images in the vertical meridian (percentage) introduced by the meridional size lenses. The visual distance was 40 cm., and the pattern consisted of two rectangles.

are illustrated graphically in figure 8. Inspection shows that the maximum sensitivity for all the visual angles within a relatively small variation is the same for the same observer and for the same pattern. In several cases perhaps a small, though not particularly significant, increase occurs.

TABLE 2.—*Summary of the Characteristic Data of Three Observers Derived from the Induced Size Effect S-Type Curves for Two Simple Types of Object Plane Patterns*

Test Pattern—Rectangles (Observer K. N. O.)							
Visual angle subtending contours	1°	2.1°	3.5°	5°	7.2°	9.2°	11.4°
Maximum sensitivity, degrees/per cent.	2.5	2.6	2.9	...	2.8	3.0	3.0
Difference in sizes of images for maximum rotation, percentage	8.0	7.0	7.2	...	6.2	5.5	5.0
Average maximum rotation, degrees	10.0	9.4	10.5	...	8.5	8.2	7.5
Center of symmetry							
Percentage	−0.9	−0.9	−1.7	...	−0.8	−1.2	−1.0
Degrees	−6.8	−7.6	−7.8	...	−6.4	−6.3	−5.4
Test Pattern—Single Contour Above (Observer K. N. O.)							
Visual angle subtending contours	1°	2.1°	3.5°	5°	7°	9°	12°
Maximum sensitivity, degree/per cent.	2.2	1.9	...	2.0	1.9	1.9	1.8
Difference in sizes of images for maximum rotation, percentage	5.0	5.5	...	5.0	4.6	4.2	3.7
Average maximum rotation, degrees	5.5	5.2	...	5.1	4.4	4.0	3.3
Center of symmetry							
Percentage	−1.1	−1.0	...	−1.0	−1.2	−1.3	−1.1
Degrees	−6.3	−6.3	...	−7.0	−7.2	−7.7	−6.8
Test Pattern—Rectangles (Observer G. S. N.)							
Visual angle subtending contours	1°	2.1°	3.5°	5°	7.2°	9.2°	11.4°
Maximum sensitivity, degrees/per cent.	1.3	1.3	1.4	1.6	1.8	1.9	1.8
Difference in sizes of images for maximum rotation, percentage	8.3	8.1	7.5	7.0	6.1	5.5	6.1
Average maximum rotation, degrees	5.0	6.0	6.6	6.6	6.3	5.3	5.6
Center of symmetry							
Percentage	−1.0	−1.0	−0.7	−1.0	−0.5	0.0	−0.6
Degrees	−1.6	−1.9	−0.9	−2.2	−2.0	−0.9	−1.6
Test Pattern—Single Contour Above (Observer G. S. N.)							
Visual angle subtending contours	1°	2°	3.5°	5°	7°	9°	12°
Maximum sensitivity, degrees/per cent.	0.8	1.0	1.3	1.0	1.4	1.0	0.8
Difference in sizes of images for maximum rotation, percentage	6.5	5.8	5.7	6.0	4.7	5.5	4.0
Average maximum rotation, degrees	2.7	2.8	3.7	3.0	3.2	2.8	1.5
Center of symmetry							
Percentage	+0.1	−0.4	−0.6	−0.8	−0.7	−1.3	−0.2
Degrees	−2.2	−3.2	−3.4	−4.3	−3.2	−3.3	−2.5
Test Pattern—Rectangles (Observer R. H. D.)							
Visual angle subtending contours	1°	2.1°	3.5°	5°	7°	9.2°	11.4°
Maximum sensitivity, degree/per cent.	1.9	2.1	2.7	2.6	2.8	2.9	2.6
Difference in sizes of images for maximum rotation, percentage	9.5	8.5	7.0	8.0	6.0	5.7	5.0
Average maximum rotation, degrees	9.3	9.0	9.7	10.5	8.4	8.3	6.6
Center of symmetry							
Percentage	−0.5	+0.1	−0.2	−0.5	0.0	0.0	−0.5
Degrees	+2.2	+1.8	+1.5	+1.6	+0.8	+0.3	−1.0

This result does not imply that the sensitivity of the retina—that is, the response of the retina per unit of area—is uniform from the fovea to the periphery. The displacement of the image of an object on the retina caused by a size lens will be proportional to the angular distance

of that point from the fixation point.[12] Therefore, the fact that the maximum sensitivity of the induced size effect, as previously defined, is apparently independent of the angular separation of the fusion contours implies that the sensitivity of the retina may decrease with the distance from the fovea to a point on the periphery. The proportional increase of the disparity of the images with the increased size of the image at least partly offsets the reduction in sensitivity. In other words, for a 1 per cent increase in the vertical meridian of the image of one eye, the resulting vertical disparity of 0.01 degree at 1 degree from the fovea and a disparity of 0.1 degree at 10 degrees from the fovea will elicit the same ocular response in an induced size effect. The decrease in retinal sensitivity expressed in the constancy of the maximum sensitivity doubtless has the same functional or structural basis as has the decrease in resolving power of the retina and the decrease in the fusion strength from the fovea to the periphery.

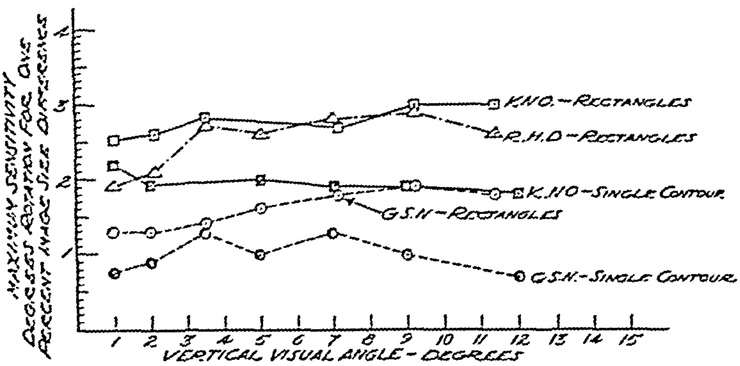

Fig. 8.—Graphic representation of the relationship derived between the maximum sensitivity to the induced size effect and the vertical visual angle subtending the fusion contours.

According to the data, however, the maximum sensitivity varies with the observer and with the pattern. Individual differences are to be expected, though the values are rather constant for the same observer. A summary of the maximum sensitivities of the different patterns for the three observers is given in table 3. This table clearly indicates that the maximum sensitivity, which reaches nearly the theoretic maximum for the full field of dots,[1] is far larger for the more complex patterns than it is for the simpler ones. This must be interpreted to mean that the retinal sensitivity is considerably higher for the more complex patterns. Accordingly, the maximum sensitivity of the effect is also larger with the more complex patterns.

12. A 10 per cent size lens, for example, placed before one eye will cause an image displacement of 0.1 degree at 1 degree from the fovea and an image displacement of 1 degree at 10 degrees from the fovea.

Difference in the Sizes of the Images in the Vertical Meridian and the Maximum Apparent Rotation at the Point of Maximum Effect: One of the most important values obtained from a given curve of induced size effect is the difference in the sizes of the images in the vertical meridian for which the maximum apparent rotation occurs. These derived values are given for each observer in the second rows of table 2 and are represented graphically in figure 9. With an increase in the separation of the contours, the curves show clearly a decrease in the values for the difference in the sizes of the images in the vertical meridian at which the maximum effect occurs; that is, the farther the fusion contours are from the fixation point, the smaller is the difference in the sizes of the images at which the maximum rotation of the test plane takes place. The relationship appears nearly linear.

TABLE 3.—*Summary of Maximum Sensitivities for Different Patterns on Object Plane* *

Pattern	Observer K. N. O.	Observer R. H. D.	Observer G. S. N.
Full field of dots	3.0	3.9	3.3
Ring of dots (5°)	3.3	3.5	2.2
Bands of dots (5°)	...	3.8	...
Black hemispheres (5°)	...	...	1.9
Average rectangles (1°-12°)	2.8	2.5	1.6
Average single contour (1°-12°)	2.0	...	1.0
Theoretic maximum †	3.6	3.9	3.8

* The values are degrees of apparent rotation for a 1 per cent difference in the sizes of the images in the vertical meridian.

† It is assumed that the value for the theoretic maximum will be equal to the theoretic sensitivity for the geometric effect, though opposite in sign.[1]

Since the maximum sensitivity is nearly constant for all visual angles subtended by the fusion contours and the difference in the sizes of the images in the vertical meridian for the maximum rotation decreases with the increase of the visual angle, it is to be expected that the magnitude of the maximum rotation also decreases with the increasing visual angles to the fusion contours. This is actually the case, as can be seen from table 2, third row, and from figure 10, in which the values of the maximum apparent rotation of the object plane are given and illustrated.

The change in the difference in sizes of the images in the vertical meridian for which the maximum effect is found and the change in the magnitude of the maximum rotation itself, both of which occur with an increase in the visual angle subtending the contours, may also be considered from the point of view of a fusion phenomenon. As suggested previously, the stimuli for fusion from a peripherally located contour becomes weaker as its distance from the fixation point is increased. Consequently, with an increase in the distance, a decrease would be

expected not only in the maximum difference in the sizes of the images in the vertical meridian but also in the magnitude of the maximum effect itself.

The differences observed between the different patterns with respect to the difference in the size of the images for the maximum effect and the maximum apparent rotation itself may be accounted for on the assumption that the induced size effect becomes smaller as the fusion

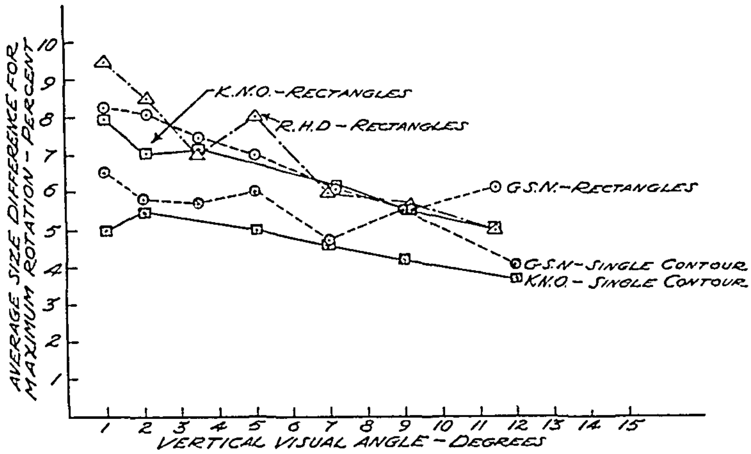

Fig. 9.—Graphic illustration of the relationship derived between the average difference in the sizes of the images in the vertical meridian for the maximum induced effect and the vertical visual angle subtended by the fusion contours.

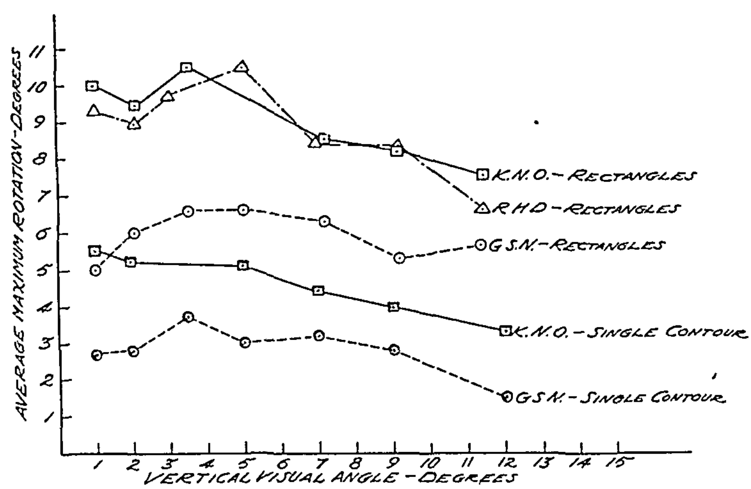

Fig. 10.—Graphic representation of the derived data showing the relationship between the average maximum rotation of the object plane in the induced size effect and the vertical visual angle subtended by the fusion contours.

stimuli become weaker. From the graphs in figures 9 and 10 it will be seen that the values for the rectangular patterns are considerably higher than those for the single contours. In considering these differences, two points must be taken into account: first, that the rectangular patterns are increased in size in proportion to their distance from the center; and, second, that the single contours were placed only above the horizontal row of dots, whereas the rectangles were placed both above and

below the dots. Thus, the fusion stimuli of the rectangles must have been more than twice as strong as that of the single contours. The weakening influence of the increasing distance of the fusion contours from the fixation point in the case of these rectangles is compensated for in part by the stimulation of a larger retinal area, both above and below the fixation point.

The values for the differences in the sizes of the images in the vertical meridian for which the maximum effect occurred may also be interpreted in terms of a limiting disparity of the retinal images. These equivalent disparities, which can easily be computed from the derived data,[13] are shown in table 4 for the various vertical visual angles subtended by the fusion contours. Figure 11 illustrates these values. It will be seen that the limiting disparity increases with increasing visual

TABLE 4.—*Equivalent Disparities of the Retinal Images for Average Difference in the Sizes of the Images in the Vertical Meridian at Which the Maximum Apparent Rotation of the Object Plane Occurred for the Various Vertical Visual Angles Subtending the Fusion Contours*

	Minutes of Arc						
	Rectangles			Single Contour			
Vertical Visual Angles of Fusable Contours, Degrees	K. N. O.	R. H. D.	G. S. N.	K. N. O.	G. S. N.	Average Rectangles	Average Single Contour
1.0	4.8	5.7	5.0	3.0	3.9	5.2	3.4
2.0	....	....	....	6.6	7.0	9.9	6.8
2.1	8.8	10.7	10.2	....	....	....	....
3.5	15.1	14.7	15.8	....	12.0	15.2	12.0
5.0	....	24.0	21.0	15.0	18.0	22.5	16.5
7.0	....	25.2	....	19.3	19.7	26.1	19.5
7.2	26.8	....	26.4	....	....	....	....
9.0	....	....	....	22.7	29.7	30.8	26.2
9.2	30.4	31.5	30.4	....	....	....	....
11.4	34.2	34.2	41.7	....	....	....	....
12.0	....	....	....	26.6	28.8	36.8	27.7

angles; this is to be expected if the retinal sensitivity to the effect decreases from the foveas to the peripheral parts of the retinas.

The data given in table 4 and illustrated in figure 11 show, however, that the limiting disparities for the maximum induced size effect are larger for the rectangular pattern than are those for the simpler single contour. This behavior is not inconsistent with the concept that the sensitivity of the retina to the effect decreases toward the periphery and that the retinal sensitivity increases with the strength of the stimulus to fusion.[14]

13. For example, the maximum effect for a visual angle of 1 degree is reached with a difference in the sizes of the images of 7 per cent (fig. 7, graph 1); hence, the vertical disparity of the retinal images is equal to 7 per cent of 1 degree, or 0.07 degree, or 4.2 minutes of arc.

14. A similar behavior of complex as against simple patterns is found in certain vertical fusion experiments involving fusion movements (Hofmann, F. B., and Bielschowsky, A.: Ueber die der Willkür entzogenen Fusionsbewegungen der Augen, Arch. f. d. ges. Physiol. **80**:6, 1900).

The limiting retinal disparities for the maximum induced size effect are of interest also from another point of view, in that they may be considered analogous to "Panum's areas of sensation," within which disparate images can be fused. The limiting disparities in the induced effect may be said to define certain "areas of induction," within which the effect increases with the vertical disparity and outside of which the effect decreases. In general, these "areas of induction" appear slightly larger than Panum's areas of binocular single vision. It was the impression of the various observers that the simple contours on the object plane appeared to "double" vertically at a difference in the sizes of the images on the vertical meridian slightly smaller than that at which the maximum apparent rotation occurred. No data exist for the vertical dimensions of Panum's areas in the peripheral parts of the retinas, with which a quantitative comparison can be made. However, since it is generally

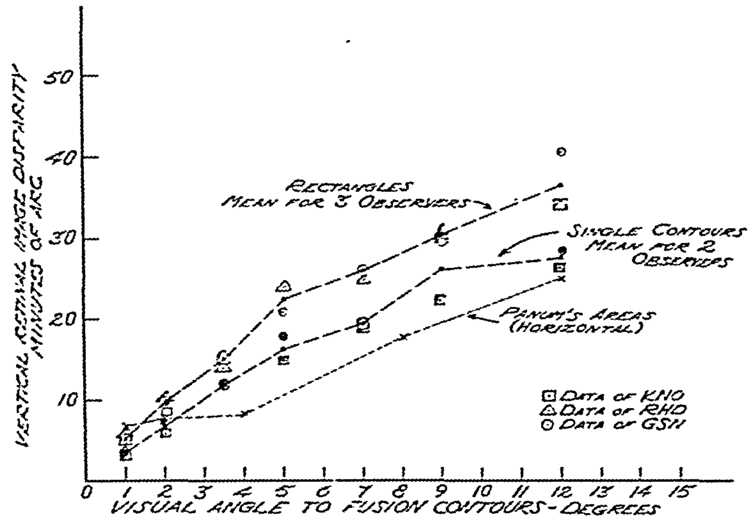

Fig. 11.—Graphic representation of the disparities of the retinal images which are derived from the data for the differences in the sizes of the images at which the maximum apparent rotation of the test plane for the different visual angles subtended by the fusion contours.

believed that their vertical dimensions are smaller than their horizontal ones, it is of interest to include in figure 11 the values for the horizontal dimensions of Panum's areas, as determined on the horopter apparatus.[15] These values permit no actual comparison but do provide a general idea as to the relative sizes of the induction areas for the two patterns and the areas of single vision.

Center of Symmetry: A comprehensive consideration of the problem raised by the fact that for some observers the center of symmetry of

15. Ogle, K. N.: An Analytical Treatment of the Longitudinal Horopter: Its Measurement and Application to Related Phenomena, Especially to the Relative Size and Shape of Ocular Images, J. Optic. Soc. America 22:665, 1932.

the induced size effect S type curve is displaced from the origin of the graphs is beyond the scope of this paper and can only be touched on. In connection with the experiments discussed here, it is important only to determine what changes occur in the displacement with the different separations of the fusion contours. The coordinates for the centers of symmetry for the foregoing series of data are given in table 2. Since the data were taken over a considerable period of time, some variation in these values might be expected. The tables, however, show no such consistent trend with increasing separations of the fusion contours within mean variations of 0.5 per cent and 1 degree. A marked trend in the displacement of the center of symmetry for the series of vertical visual angles might indicate some asymmetry of the retinal images. The displacement of the center of symmetry itself suggests an initial inequality in the relative sizes of the ocular images of the two eyes.

COMMENT

Certain fundamental facts, based on the data of three observers, have been brought out in the experiments described in the foregoing pages.

The induced size effect does not occur unless there are present in the binocular visual field at least two vertically separated contours which will exert a vertical fusion innervation. The magnitude of the effect apparently depends on the number and the orientation of the contours seen binocularly in the field of view.

With the number of contours restricted (though for some observers, even this restriction is unnecessary), the effect reaches a definite maximum for specific differences in the sizes of the images in the vertical meridian. When these differences increase still further, the effect decreases slowly. This fact indicates that the induced size effect rests on a physiologic basis.

In considering the various aspects of the foregoing data, it appears that the induced size effect may be the response to the effort of fusing two vertically unequal disparate retinal images. The strength of the response will depend then on the intensity of the stimuli for that fusion innervation. It appears also that the sensitivity to such stimuli decreases toward the peripheral parts of the retinas.

With sufficient vertical fusion stimuli in the binocular visual field, an increase in the disparities of the retinal images, introduced by size lenses, results in an increase of the induced size effect up to a certain maximum, beyond which a further increase of the disparities results in a gradual decrease of the effect. On the basis of the foregoing hypothesis, the strength exerted by the fusion stimuli from supramaximal disparities decreases, and a smaller induced effect results.

Similarly, this hypothesis explains the fact that the difference in the sizes of the images in the vertical meridian for the maximum induced size effect and the magnitude of this effect itself vary with different patterns and with the angular vertical distance of the contours from the fixation point. In general, a greater effect is obtained with patterns which present a greater fusion stimulus. Within certain limits, the greater the number of contours and the closer these are to the fixation point, the greater becomes the difference in the sizes of the images for the maximum effect and the magnitude of the effect itself.

Certain aspects in this consideration deserve special attention. For example, the fact that the maximum sensitivity, defined as the magnitude of rotation of the field for a 1 per cent difference in the size of the images in the vertical meridian in the vicinity of the center of symmetry, remains nearly constant for different separations of the vertical fusion contours. The constancy of the maximum sensitivity of the effect may be understood in terms of retinal sensitivity, if the sensitivity for the fusion of disparate images is assumed to decrease from the fovea to the periphery.[16] In these experiments such a decrease of sensitivity tends to be compensated for by the increase of the disparity of the retinal images toward the periphery caused by the size lenses. However, on this assumption, the vertical disparity of the images for which the maximum induced size effect occurs, as well as the magnitude of the maximum effect itself, must also decrease with increasing separations of the vertical fusion contours.

It is of special interest, also, to interpret the difference in the sizes of the images in the vertical meridian for the maximum induced size effect as a limiting disparity of the retinal image. On the basis of the data, it follows, first, that these limiting disparities increase toward the periphery of the retinas; and, second, that they are larger for the more complex patterns. The first fact might be expected, on the basis of the hypothesis that the retinal sensitivity gradually decreases toward the periphery of the retinas. The second fact might also be expected in that a greater response probably would occur to the stronger stimuli that arise from the more complex contour pattern. Thus, a larger disparity of the images, i. e., a greater retinal area, must be involved before the maximum effect will occur for increasing separations of the vertical contours. These limiting vertical disparities may be considered as retinal areas, in the sense of Panum's areas of binocular single vision. The existence of such "areas of induction" can be postulated, but on the basis of the foregoing data they appear unstable, since their magnitudes vary with the nature of the test pattern.

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16. Such an assumption is justified on other grounds, especially in view of the results of certain fusion movement experiments (Burian[8]).

As suggested in the preceding paper,[1] the induced size effect itself can be explained on the hypothesis that a compensatory change takes place in the relative sizes of the ocular images. So far as binocular depth perception is concerned, this theory assumes that the ocular processes can compensate for the difference in the sizes of the images in the vertical meridian which has been artificially introduced by size lenses; but such a compensation can be made only as an overall change in size, or as a meridional increase in the one meridian accompanied by a decrease of the image in the other meridian.[17] Such an image compensation obviously leaves a residual horizontal difference in the relative sizes of the ocular images, and the apparent rotation of the binocular field would occur, as in the geometric effect, from this horizontal difference. On the whole, such a hypothesis is consistent with the foregoing data and their interpretation, for the compensatory process would be the response to the fusion stimuli exerted by vertically disparate images. The fact that the maximum sensitivity of the effect for a given pattern is the same for all separations of the vertical contours is easily accounted for. Within relatively small differences in the sizes of the images in the vertical diameter, the compensatory mechanism responds to the same extent in compensating for the vertical disparity, irrespective of where the images lie. An increase in the number of contours would increase the strength of the innervation to the compensatory mechanism. Moreover, a maximum effect will simply indicate a limitation of the compensation processes. Beyond the maxima, there would be a continuous attempt to compensate for the vertical disparity, though the influence of the fusion patterns becomes weaker with the larger disparities. To explain the fact that the differences in the sizes of the images for the maximum effect vary with the angular distance of the contours in peripheral vision, it must be assumed that the limits of the compensating process vary with the retinal distances from the foveas.

The conception of a compensatory process, of course, is only a way of describing the phenomena observed. No physiologic or anatomic basis for such a process is known at present.

SUMMARY

With simplified fusion patterns it is possible to ascertain the following facts concerning the induced size effect:

1. The introduction of a difference in the sizes of the images in the vertical meridian does not cause an induced size effect unless there are at least two separated vertical contours in the binocular visual field.

17. This is true in the sense that a ribbon of rubber when stretched contracts in the direction at right angles to the direction of the stretching.

2. This difference in the sizes of the images for the maximum induced size effect decreases with increasing separations of the fusion contours.

3. The magnitude of the maximum induced size effect decreases with increasing angular distances of the fusion contours from the fixation point.

4. The maximum sensitivity (the apparent rotation of the binocular visual field for a 1 per cent difference in the sizes of the images at the center of symmetry) varies with the observers and with the patterns but is nearly constant for different visual angles subtended by the fusion contours.

The experimental findings are discussed from the point of view of the strength of fusion stimuli for disparity of the retinal images and of a hypothetic effective change in the sizes of the ocular images.

CONGENITAL TYPE OF ENDOTHELIAL DYSTROPHY

FREDERICK H. THEODORE, M.D.
NEW YORK

Endothelial dystrophy, or cornea guttata, is generally considered at the present time to be a degenerative senile or presenile condition which may progress and later predispose to epithelial changes, notably the epithelial dystrophy of Fuchs. This concept, which is the only one held by all English and American contributors to the subject, is based on the work of Vogt and of Graves. Even the recent textbook by Duke-Elder [1] shares this point of view and, like other texts, in an apparently complete discussion omits any reference to the original describers, first Koeppe and later Staehli, and fails to mention several case reports which point to a congenital basis for some of these cases. Because of this misconception, the condition in the cases reported here was at first unclassified and, in fact, considered hitherto undescribed. It was only after a fairly complete review of the literature that its nature was understood and that it was realized that there might be several types of endothelial dystrophy.

REPORT OF CASES

CASE 1.—Sylvia L., a girl of 18, was observed at birth to have "cloudy, gray" corneas. At the age of 3 months, on Nov. 28, 1919, she was taken to the clinic of the Herman Knapp Memorial Eye Hospital, where bilateral deep corneal opacities were noted, but the age of the patient precluded more detailed study. Subsequently, at several clinics in the city of New York, she was given glasses because of poor vision, but these never brought about any significant improvement. On April 12, 1938, she returned to the Herman Knapp Memorial Eye Hospital after an absence of eighteen years, hoping to improve her vision with new glasses.

Ocular Examination.—The vision in the right eye was 20/70 — 2; it improved to 20/70 + 1 with a + 0.75 sph. — 1.25 cyl., axis 105. The vision in the left eye was 20/80; it improved to 20/60 — 1 with a — 0.50 sph. — 2.50 cyl., axis 55.

The entire ocular examination gave negative results except for the cornea of each eye and the anterior capsule of the lens in the left eye. The media and the fundi showed no abnormalities or evidences of old inflammation.

The cornea of each eye was normal in size and shape. There was no impairment of sensation. Grossly, each appeared somewhat cloudy. On slit lamp examina-

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From the Herman Knapp Memorial Eye Hospital, clinic of Dr. J. M. Houlahan.

Presented before the Section of Ophthalmology of the New York Academy of Medicine, Dec. 19, 1938.

1. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 2006.

tion the epithelium and Bowman's membrane were entirely normal, as was the anterior two thirds of the substantia propria. Beginning in the posterior third, the stroma became increasingly gray and relucent in a suggestively striated manner till Descemet's membrane was reached. No evidence of past or present vascularization was seen anywhere. The posterior surface of the cornea presented a most striking picture. On direct illumination it was seen to be studded with large, irregularly-sized, round and elliptic clear structures, which were more numerous in the pupillary zone. These were also present peripherally up to the limbus (fig.1). When they were observed under low power magnification the first impression was that they resembled "Swiss cheese," but closer study revealed the clear structures to be nodules or thickened depressions rather than vacuoles. Indeed, it was easy to see when the beam was narrowed that at least the larger depressions had a definite convexity toward the examiner and encroached on the stroma. The posterior boundaries of what appeared to be nodules could not be

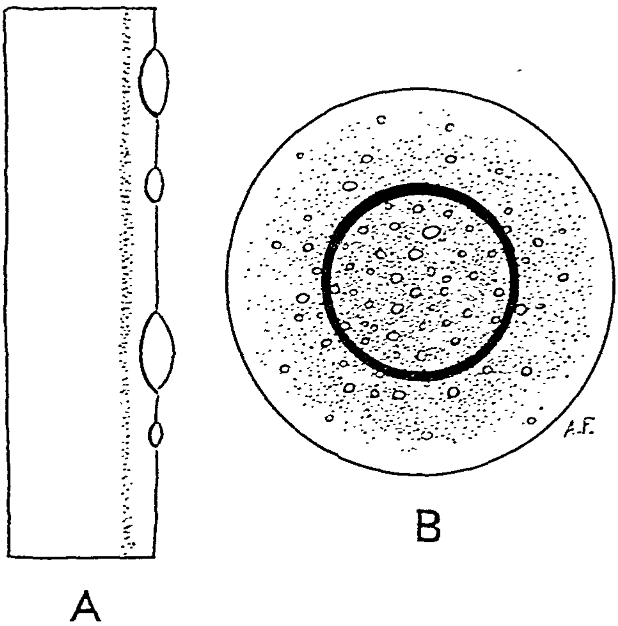

Fig. 1.—Diagrammatic sketch of the lesions in case 1.

seen clearly. The largest of these nodules measured 0.75 mm. in diameter; the average measurement was about 0.2 mm. (fig. 2). The markings of the adjacent stroma were rather relucent and accentuated the clearcut margins. On retro-illumination the nodules appeared as empty spaces. Direct illumination in the zone of specular reflection revealed the endothelium to be completely absent wherever a nodule was present but entirely normal in most other areas (fig. 3). In some places, however, normal endothelium merged into distorted portions, forming whorls of irregular, golden reflections having a mottled pattern. No deposits of any nature were found on the posterior endothelial surface of each eye.

As far as could be determined, it appeared that the structures were excrescences of Descemet's membrane, similar in appearance to Henle's warts but much larger.

On the anterior capsule of the lens of the left eye were a few scattered clusters of star-shaped and v-shaped brown pigment of the type considered congenital.

General Examination.—General physical examination gave essentially negative results. A tendency to a male type of escutcheon was noted. Although the patient

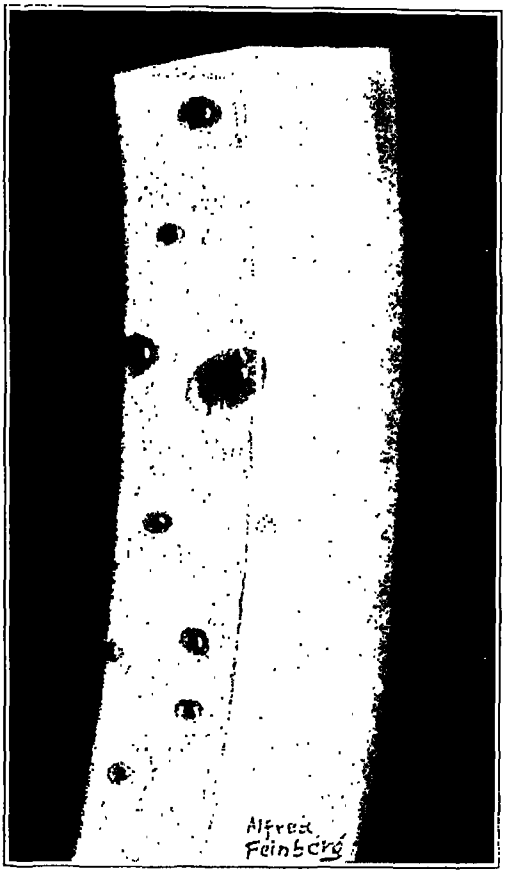

Fig. 2.—Slit lamp appearance on direct illumination in case 1; × 16.

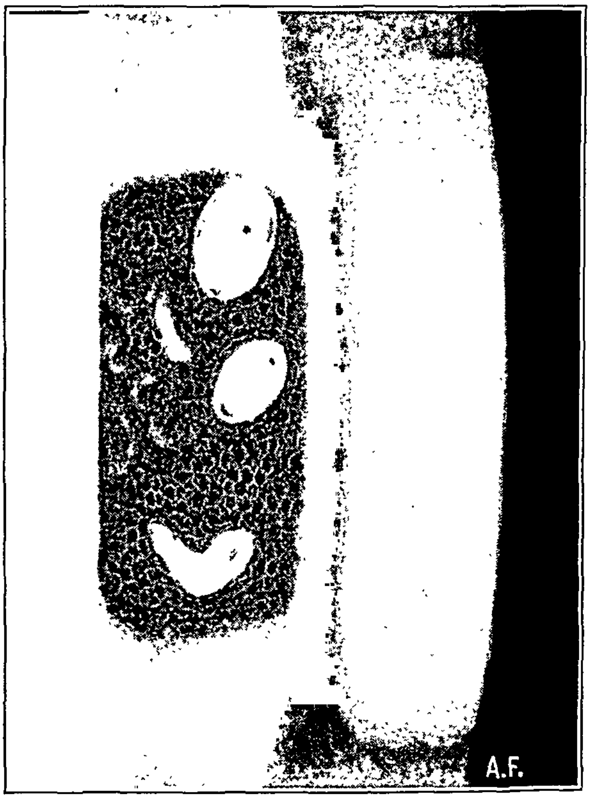

Fig. 3.—Slit lamp appearance on specular reflection in case 1; × 31.

had once been told that she had a pilonidal sinus, no evidence was found on this examination. No other congenital abnormalities were found. In the fall of 1936 she had undergone investigations because of scanty and infrequent menstrual periods. The examination included a study of the endocrine system, which revealed no abnormalities other than a slightly lowered basal metabolic rate, — 16. In the course of a year her periods became practically normal without any treatment. The Wassermann reaction of the blood was negative.

All of the patient's immediate family were then examined. A sister and two brothers were found to be essentially healthy, and their eyes proved to be completely normal. The mother, who had had trouble with her eyes as a child, showed evidences of old eczematous keratitis in each eye. In the central two thirds of each cornea was a superficial opacity. The posterior part of the cornea was entirely normal except for some scattered deposits of pigment on the endothelium, which itself was intact.

Examination of the father, however, requires fuller description.

CASE 2.—Jacob L., 53 years old, the father of the patient in case 1, had never had any trouble with his eyes until a few years prior to examination, when he had begun to have presbyopic symptoms. He had always thought that he could see well, but his visual acuity had never been tested previously.

Ocular Examination.—The vision in the right eye was 20/60; it improved to 20/30 — 1 with a — 0.50 sph. — 0.50 cyl., axis 30. In the left eye it was 20/30 —1 and was only improved to 20/30 + 1 with a — 0.25 sph. — 0.25 cyl., axis 145. With a + 2.25 addition for near vision in each eye he saw the second smallest test type.

On examination each eye was entirely normal except for the cornea. Here, a picture similar to that of his daughter's was present in each eye (fig. 4). Except for a small, superficial corneal scar in the left eye, probably due to an old foreign body, the changes were likewise in the posterior part of the cornea. Parenchymal clouding began diffusely in the posterior third and in the lower portion was accentuated to form an opaque, triangular, wedge-shaped area with its apex toward the center of the cornea, quite similar in appearance to embryotoxon. No vascularization was present. Descemet's membrane was similarly studded with excrescences, as in case 1, but here they were much smaller and tended to be grouped. They measured from 0.08 to 0.12 mm. in diameter on the average. A few measured 0.2 mm. Some showed an anterior convexity. The endothelium was absent behind these areas but intact between them. In the left eye a small amount of brown pigment was present on the endothelium in the center of the cornea, forming a fine horizontal line. The corneal sensation was normal.

General Examination.—General physical examination, as in the case of the daughter, gave negative results. The Wassermann reaction of the blood was negative.

This second case confirmed the congenital character of the anomaly and prompted further study of the father's family.[2] His father, two

2. Dr. Benjamin Sachs, of Boston, allowed me to use his office facilities to study this group of patients.

brothers, two sisters and their six children were found to be completely normal on slit lamp examination. The mother, however, showed corneal changes, which are described in case 3.

CASE 3.—Jennie L., a 77 year old housewife, the grandmother of the patient in case 1, had always been healthy until the two years prior to examination, when she had begun to fail in strength. She had never had any miscarriages. She recently suffered from severe headaches, which probably were due to hypertension. Her blood pressure was 230 systolic and 100 diastolic. Her eyes had never bothered her.

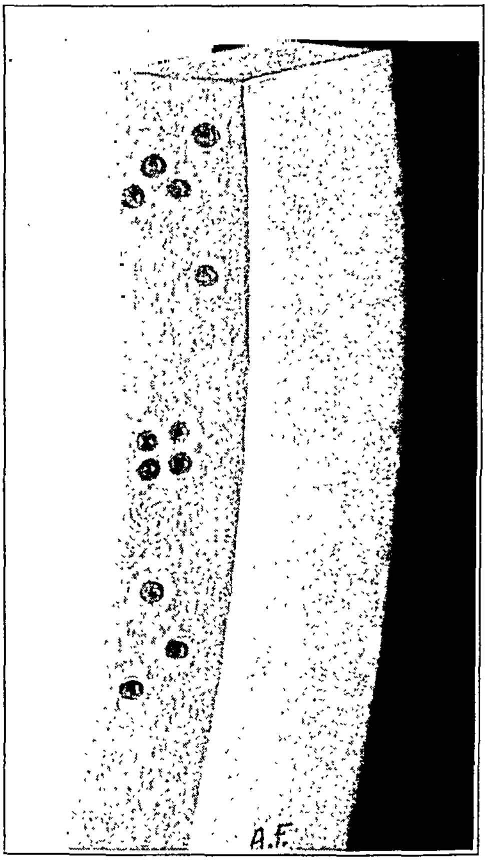

Fig. 4.—Slit lamp appearance on direct illumination in case 2; × 24.

Ocular Examination.—The vision in the right eye was 20/100 and could be improved to only 20/70 with a —75 sph. In the left eye vision was 20/200; it improved to 20/40 + 2 with a + 1.50 sph. — 3.00 cyl., axis 90.

Except for a moderately advanced senile nuclear cataract in the right eye, which caused the visual impairment, and moderate arteriosclerotic changes in the fundus, the only significant ocular findings were corneal. These were bilateral.

Here, as in cases 1 and 2, the pathologic process commenced posteriorly, the epithelium and practically the entire stroma being normal. A little diffuse stippled clouding began just anterior to Descemet's membrane. Scattered, rounded excrescences were found all over Descemet's membrane but were especially noticeable below the center, where they were slightly larger. In the center itself, dis-

tortions of the endothelium, as in case 1, were particularly noticeable, with whorls seen in specular reflection. In this case in contradistinction to the other 2 cases, considerable golden brown pigment was found on and in the endothelium, some of which, at least, appeared intracellular. The nodules were not measured, as no ruled eye piece was available, but they seemed to be somewhat smaller than those in case 2 and much smaller than those in case 1 and were less numerous. The sensibility was unimpaired.

To recapitulate, a daughter, her father and her grandmother, three generations in all, were found to have a bilateral, nodular, anomalous disturbance of Descemet's membrane and endothelium, with impairment of vision proportionate to the degree of pathologic involvement. With each succeeding generation the condition became worse. Except for an increased relucency of the adjacent stroma, the remainder of the ocular examination of each patient gave negative results. General physical examination gave essentially negative results, and no degenerative familial stigmas were present.

REVIEW OF THE LITERATURE

Although it is ignored in the leading textbooks and by most other authors on this subject, the first description of endothelial dystrophy was made by Koeppe [3] in 1916. To the curious structures that studded the posterior corneal surface bilaterally in his six cases, he gave the name of *Dellenbildungen*, translated as "facets" or "pits." The convexity of these structures was toward the observer. The cases were excellently described, and there can be no question that he was dealing with the subject under discussion. All his patients had subnormal vision, which could not be completely corrected. His youngest patient was 25 years old, and the average age of the group was about 35 to 40 years. By 1920 [4] he had collected 18 cases. Sixteen of the patients had normal eyes except for the corneal lesion. Of the remaining 2, 1 had iridocyclitis in one eye only, but both eyes had the "pitting." The other patient had chronic simple glaucoma. The patients stated that their vision had been the same since youth. Therefore, in view of the absence of other pathologic changes, Koeppe concluded that the condition was congenital and was due to an anomaly in the development of the posterior corneal surface.

The second author on this subject was Staehli,[5] who in 1920 described 12 cases in older persons. He spoke of a "bedewing" of the posterior

3. Koeppe, L.: Klinische Beobachtungen mit der Nernst Spaltlampe und dem Hornhautmikroskop, Arch. f. Ophth. **91**:375, 1916.

4. Koeppe, L.: Die Mikroskopie des lebenden Auges, Berlin, Julius Springer, 1920, p. 130.

5. Staehli, J.: Ueber eine besondere Form von Betauung der Kornealrückfläche, Klin. Monatsbl. f. Augenh. **65**:106, 1920.

corneal surface as a manifestation of senile degeneration and named it "dew-drop endothelium." While his descriptions cannot compare with Koeppe's he was the first to point out that the patients were prone to see colored rings around lights, owing to refraction phenomena. Also in 1920 Kraupa [6] wrote a paper associating posterior corneal pigmentation with epithelial dystrophy.

In 1921 Vogt,[7] who usually is credited with priority, reported his observation of this condition, which he later called cornea guttata. He correctly interpreted the excrescences to be similar anatomically to Hassall-Henle bodies and considered the process a senile change. In 1922 Moeschler [8] in a study of posterior corneal pigmentation considered both this and cornea guttata to be due to senile changes. In 1923 Kraupa [9] first mentioned the relationship of the endothelial changes to the epithelial dystrophy of Fuchs.

Undoubtedly the fullest and best anatomic description of the syndrome was published by Graves [10] in 1924. For the first time adequate illustrations appeared. On the basis of the age incidence, he considered the condition a presenile or senile phenomenon. In rapid succession a series of papers by Kirby,[11] who first called the condition "endothelial dystrophy," Triebenstein,[12] who independently gave it the same name, H. and J. S. Friedenwald,[13] Gifford [14] and Sallmann [15] appeared. Influ-

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6. Kraupa, E.: Pigmentierung der Hornhauthinterfläche bei "Dystrophia epithelialis (Fuchs)," Ztschr. f. Augenh. **44**:247, 1920.

7. Vogt, A.: Weitere Ergebnisse der Spaltlampenmikroskopie des vordern Bulbuschnittes, Arch. f. Ophth. **106**:63, 1921.

8. Moeschler, H.: Untersuchungen über Pigmentierung der Hornhautrückfläche bei 395 am Spaltlampenmikroskop untersuchten Augen gesunder Personen, Ztschr. f. Augenh. **48**:195, 1922.

9. Kraupa, E.: Die familiären degenerativen Hornhautveränderungen (neurotische Dystrophie und Ichthyosis corneae) in System der sogenannten Dystrophien der Hornhaut, Klin. Monatsbl. f. Augenh. **70**:396, 1923.

10. Graves, B.: A Bilateral Chronic Affection of the Endothelial Face of the Cornea, Brit. J. Ophth. **8**:503, 1924.

11. Kirby, D. B.: Excrescences of the Central Area of Descemet's Membrane, Arch. Ophth. **54**:588 1925.

12. Triebenstein, O.: Veränderung an der Hornhauthinterfläche, Klin. Monatsbl. f. Augenh. **74**:777, 1925.

13. Friedenwald, H., and. Friedenwald, J. S.: Epithelial Dystrophy of the Cornea, Brit. J. Ophth. **9**:14, 1925.

14. Gifford, S. R.: Epithelial Dystrophy and Erosion of the Cornea, Arch. Ophth. **54**:217, 1925; Epithelial Dystrophy of the Cornea and Its Relationship to Endothelial Dystrophy, Am. J. Ophth. **9**:81, 1926; The Mild Form of Epithelial Dystrophy of the Cornea, Arch. Ophth. **7**:18 (Jan.) 1932.

15. Sallmann, L.: Ein Fall von Dystrophia endothelialis corneae, Klin. Monatsbl. f. Augenh. **75**:778, 1925; Ueber eine dystrophische Endothelveränderung und Pigmentierung der Hornhautrückfläche, Ztschr. f. Augenh. **58**:348, 1926.

enced by Vogt, they all considered the syndrome to be degenerative, although Kirby mentioned the possibility of a hereditary or familial factor, and the majority of the authors were concerned mainly with the possible relationship to the epithelial dystrophy of Fuchs. By this time the association of endothelial dystrophy with high myopia and glaucoma had been noted, and in 1930 Sallmann [16] published his observation that in 3 cases associated with glaucoma epithelial erosions occurred when he took the tension, indicating the presence of a diseased or more readily damaged epithelium. In this connection Peter's [17] article in 1931, in which he reported that he found signs of cataract in every one of 22 patients, is interesting, as he pointed out that patients with endothelial dystrophy are poor surgical risks.

In 1930 the results of the first histologic study of cornea guttata were published by Vogt.[18] In the second edition of his atlas [19] are excellent illustrations of the biomicroscopic picture and of the histologic specimens. Vogt's observations were confirmed by Goar [20] in 1934. Essentially, the changes consist of irregular thickenings and excrescences of Descemet's membrane. The endothelial cells over these nodules are thinned, even absent, and the cytoplasm contains many pigment granules. The excrescences have staining properties identical with those of Descemet's membrane. Goar expressed the belief that the condition occurs in about 6 per cent of adults, in women four times as often as in men. In 1934, Vogt [21] observed similar changes histologically in another case, and, in addition, it was found that the endothelium was uneven in areas in which there were no excrescences, which would explain the whorl-like appearance seen on specular reflection in the cases described in this paper.

In all the aforementioned reports, with the exception of Koeppe's contributions, it is uniformly agreed that the condition is a senile degenerative change. This conception was challenged by Kraupa,[22] who in

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16. Sallmann, L.: Zur klinischen Bedeutung einigen Spaltlampenbefunde an Hornhaut und Linse, Arch. f. Ophth. **125**:62, 1930.

17. Peter, L. C.: Dystrophy of the Corneal Endothelium: Its Recognition and Clinical Significance, Arch. Ophth. **6**:817 (Dec.) 1931.

18. Vogt, A.: Der histologische Befund bei Cornea guttata, Klin. Monatsbl. f. Augenh. **85**:282, 1930.

19. Vogt, A.: Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges, ed. 2, Berlin, Julius Springer, 1930, vol. 1, p. 99.

20. Goar, E. L.: Dystrophy of the Corneal Endothelium, Am. J. Ophth. **17**: 215, 1934.

21. Vogt, A.: Neue histologische Befunde bei Cornea guttata, Ztschr. f. Augenh. **84**:21, 1934.

22. Kraupa, E.: Ueber Epitheldystrophien der Hornhaut, Wien. klin. Wchnschr. **45**:539, 1932.

1932 reported endothelial dystrophy in three generations, a grandmother of 70, an albinotic daughter of 40 and an albinotic granddaughter of 10.[23] The woman of 40 had the most pronounced changes and pigmentation, while the young girl had less involvement. A few months later Freudenthal [24] reported the condition in a boy of 14 and in his father, aged 42. Both showed a delicate, deep, diffuse parenchymal clouding as well. Because the son showed general changes suggestive of hypothyroidism, although an unsatisfactory basal metabolic test showed the metabolism to be + 20, and the father evidence of general glandular dysfunction, Freudenthal suggested a relationship to general degenerative states. Although the serologic reactions of the blood were negative, he thought that hereditary syphilis might be a factor in causing this endocrine depression, because the father's sister had a 2 plus Wassermann reaction of the blood and an old interstitial keratitis.

The entire subject of epithelial and endothelial dystrophy was then reviewed by Kraupa [25] in 1934. He reaffirmed his contention that endothelial dystrophy is definitely hereditary if not congenital. He offered, in addition, a classification of the epithelial dystrophies which should be mentioned. The outstanding point he made in this connection was that Fuchs' epithelial dystrophy is quite distinct from other types and consists of a general neurotropic disturbance of the entire cornea. According to him, the epithelial changes for which the condition is named are only part of the picture, just one symptom, and the endothelial changes do not occur prior to the rest of the picture in these rare cases, as he had previously thought, but are another part of the general disturbance. One infers from his paper that he believed that the epithelial changes which come on late in the course of endothelial dystrophy belong to an entirely different type of dystrophy. He did not attribute any hereditary character to Fuchs' dystrophy and implied that persons with familial or hereditary histories of dystrophy have the *disjunctio epithelii* of von Szily, which is often misinterpreted as Fuchs' type, or a new type he described under the name of "juvenile epithelial dystrophy," which is of a familial nature. According to Kraupa, these four types are completely confused in the literature, and the condition in many cases has been called Fuchs' dystrophy through error.

23. The actual description of these cases is found in a later (1934) paper in which Kraupa claims priority over Freudenthal.

24. Freudenthal, E.: Ueber zwei Fälle von familiärer Endotheldystrophie der Hornhaut bei Vorhandensein allgemein degenerativer Veränderungen, Ztschr. f. Augenh. **78**:224, 1932.

25. Kraupa, E.: Ueber Epithel- und Endotheldystrophien, Ztschr. f. Augenh. **83**:179, 1934.

Further reports mentioning the relationship of epithelial to endothelial dystrophy were made by Biozzi [26] and Sala [27]. In 1935 Horniker [28] in a study of patients with endothelial dystrophy or posterior corneal pigmentation commented on the frequency of the conditions and concluded that they are due to a disturbance of the neurovegetative system and are manifestations of a special angioneurotic constitution. He found patients with circulatory disturbances, neuroarthritis, hemicrania and scintillating scotomas. The allergic character he thought proved by the occurrence of urticaria, hay fever, mucous colitis, angina pectoris (!) and asthma in some of these patients.

The most recent paper to appear on the subject is that of Motolese [29] in 1938. He described a case of endothelial dystrophy in a man with pulmonary tuberculosis. The father had syphilis, and some siblings had pulmonary tuberculosis. In the course of six years a "typical" Fuchs' epithelial dystrophy occurred in each eye. The vision in one eye, however, improved during this period. The other eye became practically blind. He expressed the belief that the corneal condition became worse as the patient's general condition became poorer. As far as the endothelial dystrophy is concerned, his failure to describe it adequately until the last examination is to be regretted. Motolese concluded that endothelial dystrophy may be found in patients of any age or perhaps congenitally and is due to a disturbance of a general nature or changes such as Horniker described. His otherwise complete paper does not dwell enough on the evidence for these statements. He also described 2 cases of familial juvenile epithelial dystrophy similar to those of Kraupa, to which group Curin [30] in 1937 had added a case of the congenital type.

COMMENT

The cases reported in this paper are notable because in three generations a similar, dystrophic condition of Descemet's membrane and of the endothelium appeared. In each succeeding generation the condition was more pronounced. There is no question of the hereditary character of the changes. Nor can there, likewise, be any reasonable doubt as to the

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26. Biozzi, G.: "Cornea guttata" e reperti associati, Arch. di ottal. **41**:357, 1934.

27. Sala, G.: Opacità a cintura e cornea guttata, Rassegna ital. d'ottal. **3**:860, 1934.

28. Horniker, E.: Le distrofie endotheliali della cornea ed il loro significato, Atti d. Cong. Soc. oftal., 1935, p. 199; abstracted, Zentralbl. f. d. ges. Ophth. **35**: 690, 1935-1936.

29. Motolese, A.: Distrofie epiteliali ed endoteliali della cornea, Boll. d'ocul. **17**:1, 1938.

30. Curin, J.: Die "juvenile Epitheldystrophie," Ztschr. f. Augenh. **91**:185, 1937.

congenital origin in the first case. The cloudiness of the cornea was noted at birth and was confirmed by ophthalmologists at the age of 3 months. The patient's vision was poor from infancy and did not change. While one cannot be as certain in the case of her father, since he was older and did not have enough visual impairment to notice it, it appears likely that he, too, was born with the condition. The triangular, wedge-like segment described had definitely the appearance of a congenital lesion. As regards the grandmother, although there was no history to go by, one can only surmise that she, too, was born with the condition.

The next question is whether the conditions in these cases belong in the category of endothelial dystrophy. In the light of what a complete review of the literature reveals about the syndrome, one is justified in classifying them as such. The character, location, and distribution of the lesions are essentially the same. In only the first case does the picture differ at all from that in the previously reported cases. The nodules (average, 0.2 mm.; some 0.75 mm. in diameter) are so much larger than usual (from 0.04 mm. to 0.08 mm.) that, at first glance, they might seem different. Actually, the difference is only quantitative.

The question of the direction of the convexity of the nodules, although apparently only a detail, gives rise to interesting speculation. The larger nodules seen in cases 1 and 2 had an anterior convexity. Vogt and Graves, who expressed the belief that the condition is senile, described the nodules as having a posterior convexity, i. e., toward the anterior chamber. This corresponds with the pathologic picture and would seem logical if the nodules appeared after the complete development of the cornea, such as after birth, since extension would most likely occur in the direction of least resistance—toward the endothelium rather than toward the tougher stroma. But if the nodules were large enough and the original disturbance occurred early enough in the embryo, the stroma might be encroached on, in fact, be partially deficient at these sites. Descemet's endothelium is present at the 20 mm. stage, and Descemet's membrane is definitely present at 76 mm. The parenchymal development is not complete till much later. Thus, it is not too difficult to imagine the nodule being present before the stroma is in a final state of development and so having an anterior convexity as well as a posterior one. In further support of this theory is the fact that in the 3 cases here presented, as well as in Freudenthal's cases of the congenital type, the posterior portion of the stroma showed clouding and diminished transparency. That this should occur when the smooth and even arrangement of the fibers of the stroma is irregularly impaired by the protuberances seems reasonable; in fact, it is even to be expected. Moreover, the increased relucency is of a striated type. Koeppe, the proponent of the congenital nature of the condition, also described an

anterior convexity to the nodules. Kraupa, who expressed the same opinion as regards the congenital nature, thought that the nodules had a posterior convexity. His own cases are not fully described, and it is possible that the nodules were small. Freudenthal omitted any mention of this detail entirely. I could not find a definite anterior convexity in my third case in which the nodules were small. Thus, it seems likely that the biconvexity of many of the nodules is a corollary to their large size and to their appearance early in embryonic life.

Another speculative point is raised by the fact that in the cases of the definitely congenital type no evidence of epithelial change occurred, even in the 77 year old woman whose case is reported here and the 70 year old female patient of Kraupa. The incidence of endothelial dystrophy is high and that of epithelial dystrophy low, and the relationship may be accidental, as some still believe; but it may be significant that in the cases reported here no relationship was found to exist, while in cases of the senile type the two conditions are sometimes found together. Motolese did not consider the condition in his case of congenital origin. Moreover, some authors, notably Vogt and Graves,[31] described the condition in their cases of pure endothelial dystrophy of the senile degenerative type as progressing in the course of time, with diminution of vision. The condition in the cases of proved congenital origin and in those cases in which one may assume the origin to be congenital seems to show no such progression, and the vision is stationary. This is what one would expect of a congenital anomaly. Moreover, in the cases reported here the youngest patient had more pathologic involvement than the oldest.

While the differences suggested here are only conjectures and not presented as valid arguments, a study of the subject leads to the conclusion that there may really be two types of endothelial dystrophy, one a senile change of frequent occurrence, sometimes associated with epithelial dystrophy, and the second, a congenital form. Similar excrescences of Bruch's membrane are known to occur in these two forms. It is unfortunate that the early work of Koeppe was so routinely disregarded by all authors, influenced as they were by Vogt's contributions, except Kraupa and Motolese. The occurrence of the congenital type cannot be doubted. But it appears rather illogical to assume, as Kraupa does, that the condition is congenital in all cases because it is in some. The great majority of the observers noted the condition in elderly persons, and while it may be true that they might have been more carefully studied than younger persons and thus the condition more readily discovered, the fact remains that most of the cases of endothelial

31. Graves, B., in Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936, p. 474.

dystrophy are found in elderly persons. Of course, had Koeppe's conception been considered, much of the work might have been done from a different point of view. However, only after large groups of children are shown to have this disturbance as frequently as their elders can the proponents of the purely congenital nature of the abnormality be truly certain. Until then, it seems safer to assume that endothelial dystrophy has at least two forms, one due to developmental changes and the other due to senile degeneration.

CONCLUSIONS

Three cases of endothelial dystrophy occurring in a daughter, her father and her grandmother are described. The condition in these cases must be classified as of congenital origin. A review of the literature reveals that some authors consider the condition to be congenital, while the great majority believe it to be a senile degenerative change. On the basis of evidence at my disposal and some apparent differences noted in the two types, it is suggested that endothelial dystrophy be considered to have two forms, one congenital and the other senile.

ADDITIONAL RESEARCH ON VERNAL CONJUNCTIVITIS

LOUIS LEHRFELD, M.D.
Attending Surgeon, Wills Hospital
AND
JEROME MILLER, M.D.
PHILADELPHIA

Vernal conjunctivitis is a specific form of ocular disease. It is seasonal in character, beginning in the spring, and is characterized clinically by itching, lacrimation, photophobia, a bloodshot appearance and a mucous discharge, often containing eosinophils. It occurs chiefly in children with a past or present history of some allergic disease and in members of families in which other allergic diseases occur. It responds symptomatically to treatment with epinephrine.

DESCRIPTION OF DISEASE

Beginning as early as the pollination period of the trees in April or later in the spring coincident with the pollination of the grasses or at any time in the late spring and early summer, vernal conjunctivitis may continue through the hot season until the first cool weather in September.

There are two distinct varieties of the disease, the limbic, or corneal, type and the lid, or palpebral, type. They differ in their appearance by reason of the anatomic locations of the lesions, the limbic type being of shorter duration, lasting only for the seasonal attack and leaving no telltale pathologic involvement, while the lid type, after one or more seasonal attacks, may continue to manifest pathologic lesions throughout the year, but with an absence of symptoms during the cold season. The lid type is always characterized by stringy, chewing-gum mucus, while in the limbic form the mucus is less viscid and much less in quantity.

The characteristics of the objective symptoms have been reported in earlier publications. Suffice it to say that the limbic type maintains itself as single, discrete, phlyctenular-like elevations at the limbus, attended by vascularization, or in the form of confluent phlyctenules, two, three or more, or a complete or incomplete ring of icing effect at the limbus.

Read in part before the Section on Ophthalmology, College of Physicians, Philadelphia, Feb. 16, 1939.

These elevations are found only in the acute stage of the disease; they reach their height in about two or three weeks and then, like the wheals of urticaria, slowly recede. Because of the hyperemia of the conjunctiva, both bulbar and palpebral, there will be noticed follicular enlargements resembling the simple type of palpebral vernal conjunctivitis. It is this manifestation coincident with the limbic type which gives the often mistaken impression that the limbic and the lid types may occur at the same time. From experience, however, the limbic type and the lid type never occur coincidentally in the same patient.

The lid types are divided into: (1) the simple follicular type, (2) the pavement epithelium (cobblestone) type and (3) the granuloma or giant cobblestone type.

For a further description, the reader is referred to a previous article by one of us (L. L.).[1]

OPHTHALMIC AND INTRADERMAL TESTS

The present study was made in an effort to determine whether or not vernal conjunctivitis is truly an atopic disorder due to pollens and other atopens or whether it is due to some extraneous nonspecific agent or to some agent with which the eye comes in contact.

The tree pollen extracts used for both the intradermal and the ophthalmic tests were those of the nine common trees—ash, beech, birch, elm, hickory, maple, oak, poplar and sycamore. Other pollens utilized were those of the grasses and weeds—timothy, plantain and ragweed. Allergens used for the intradermal tests consisted of the common inhalant group and foods, such as vegetables, fruits, cereals, fish, meat, dairy products, nuts and spices.

Many patients referred to us were excluded from the present series, as they constituted seasonal hay fever sufferers with ocular symptoms predominating. Physical examination failed to reveal any endocrine disorders or physical maldevelopments.

Diagnosis was based on a careful detailed history, personal and family histories for allergy, eosinophils in the discharge from the eye and in the blood, seasonal incidence, positive reactions to the ophthalmic and intradermal tests and the clinical ocular findings.

The intracutaneous method of testing was employed in this series of cases. We made use of the intradermal rather than the scratch method, it being more accurate and sensitive, and we preferred obtaining a false positive reaction rather than miss any reaction that we otherwise would if the scratch test were used. All extracts were freshly prepared, and their strength was based on the total nitrogen content as determined

1. Lehrfeld, L.: Vernal Conjunctivitis: Observations on Eighty-Seven Cases at the Wills Hospital (1929-1931), Arch. Ophth. 8:380-404 (Sept.) 1932.

by the Kjeldhal method. The tests were performed on the outer aspect of the arm, which was first cleansed with 70 per cent alcohol. A 1 cc. syringe graduated by hundredths was used. The needles were 26 gage and 3/8 inch (0.9 cm.) in length. All intradermal injections were made as superficially as possible. Enough of the antigen was introduced to produce a wheal, which in no case exceeded 1 mm. Controls of buffered saline solution were used in all cases. The reactions were read in from ten to fifteen minutes after injection and classified into marked, moderate, slight, doubtful and negative.

DEGREES OF POSITIVE REACTIONS

A marked reaction was one in which a large wheal was obtained, usually of 10 mm. or more, with pseudopod formation and projection into the surrounding zone of erythema. This reaction was usually accompanied by itching. A moderate reaction consisted of a definite increase in the size of the wheal but without pseudopod formation. There was a definite zone of erythema, and itching was sometimes present. A slight reaction consisted of a slight increase in the size of the wheal, with a surrounding zone of erythema. A doubtful reaction was one that was slightly larger than the control reaction. Patients with doubtful reactions were always retested on another day. The sensitivity of the skin was always taken into consideration. No delayed reactions were considered.

Keeping in mind that a positive reaction to the intradermal test is only an index of cutaneous hypersensitivity, we attempted to correlate these findings with the reaction to the ophthalmic test. This test consists of the introduction into the lower cul-de-sac of dry pollen or antigen by means of the pointed end of a toothpick. The reactions thus obtained are read in from three to five minutes and again at the end of a ten minute period. A positive reaction consisted of intense immediate congestion of the bulbar and the palpebral conjunctiva and of the caruncle. All positive reactions were controlled by the instillation of a solution of epinephrine hydrochloride in a concentration of 1:1,000. Pine pollen was used as a control. (Pine is not an excitant.)

Passive transfer tests were not done in the present series, so that the presence of reagins could not be determined. Conjunctival smears and blood examinations were made for eosinophils. Patch tests were performed on a small series of the group but were persistently negative.

RESULTS

Of the 120 patients who were studied, 40 of 109 gave a family history of allergy. Thirty-seven of these had a history of allergy on one side of the family and 3 had a history of allergy on both sides of the family. There were 69 with no history of allergy. While this figure

may be slightly lower than that for patients with hay fever and asthma, it stresses the importance of the influence of heredity in vernal conjunctivitis (table 1).

The individual atopic diseases affecting the subjects studied included asthma, hay fever, atopic eczema, migraine and urticaria. Thirty-six of the 92 patients under observation gave a history of atopic disease (table 2).

The ages for this series of patients varied from 1 year to 56 years. There were 82 males as compared to 38 females. The group included 30 Negroes. Sixty-five of the 120 patients were under 10 years of age, while 48 were between the ages of 11 and 30 years. Here again the age incidence is highly significant, for it is commonly agreed that the greater

TABLE 1.—*Family History of Atopic Disease*

	Number of Males	Number of Females	Total Number
Unilateral			
(a) Maternal	16	9	25
(b) Paternal	10	2	12
Bilateral	2	1	3
Negative	46	23	69

TABLE 2.—*Personal History of Atopic Disease*

Disease	Number of Males	Number of Females	Total Number
Asthma	7	1	8
Hay fever	6	2	8
Atopic eczema	5	3	8
Urticaria	8	2	10
Migraine	1	1	2
Negative	..	..	58

the inheritance the earlier the age of onset of allergic symptoms. The sex incidence is again similar to that for other allergic conditions, for which it is found that in the first decade the males usually predominate over the females.

There were 31 cases of the lid type and 89 of the limbic type. In a previous report by one of us (L. L.) a little more than half of the total of 87 cases were of the lid type and the remainder were of the limbic type. The preponderance of males over females and of one type as compared with the other type depends on the season under study. It is the experience of one of us (L. L.) that considering several seasons jointly, the lid type is far in excess of the limbic type. The explanation is that the lid type has a much longer duration in number of seasons than the limbic type. It is common to see an accumulated group of cases of the lid type over many seasons, while the number of cases of the limbic type in which the condition is of one, two or three seasons'

duration shows a tendency to wane in comparison with the other type. Our joint experience, however, indicates that there are more male patients than females. The Negro subjects were found to manifest the limbic type only (table 3).

Of 46 patients tested by the intradermal method with the inhalant group 44 gave positive reactions to one or more agents of the group (table 4).

TABLE 3.—*Incidence of Vernal Conjunctivitis—Sex, Age and Race*

	No.	0-5	6-10	11-15	16-20	21-30	31-40	41-50	51-Over	Negro	White
Both types											
Male........	82	26	24	16	5	10	1	0	0	21	61
Female.....	38	7	8	6	7	4	4	1	1	9	29
Total.....	120	33	32	22	12	14	5	1	1	30	90
Limbic type											
Male........	57	18	17	10	3	8	1	0	0	21	36
Female.....	32	5	6	6	5	4	4	1	1	9	23
Total.....	89	23	23	16	8	12	5	1	1	30	59
Lid type											
Male........	25	8	7	6	2	2	0	0	0	0	25
Female.....	6	2	2	0	2	0	0	0	0	0	6
Total.....	31	10	9	6	4	2	0	0	0	0	31

TABLE 4.—*Results of Intradermal Tests with Inhalant Group*

	Number of Tests	Number of Positive Tests	Number of Persons Tested	Number of Reactors
Lid type......................	194	71	14	13
Limbic type..................	403	123	32	31
Total.....................	597	194	46	44

TABLE 5.—*Results of Intradermal Tests with Grasses and Weeds*

	Number of Tests	Number of Positive Tests	Number of Persons Tested	Number of Reactors
Lid type..........	126	37	21	14
Limbic type....	362	72	61	35
Total...................	488	109	82	49

Eighty-two patients were tested with the grasses and weeds, with 49 positive reactors (table 5).

Of the 46 patients tested with the pollens of the trees, 34 reacted positively (table 6).

Of the inhalant group, the most frequent antigens causing a positive reaction were dust and feathers. Thirty-three of 40 persons reacted to house dust; 15 of 36, to feathers. Next in frequency were silk, tobacco,

wool, goat epithelium, orris root, kapok and Pyrethrum. Fifty-three patients were also tested with the combined food and inhalant group, of which 52 showed positive reactions (table 7).

TABLE 6.—*Results of Intradermal Tests with the Pollens of Trees*

	Number of Persons Tested	Number of Reactors	Number of Tests	Number of Positive Tests
	0.1 Mg. of Total Nitrogen			
Lid type	5	5	45	25
Limbic type	15	14	135	64
Total	20	19	180	89
	0.01 Mg. of Total Nitrogen			
Lid type	13	9	117	36
Limbic type	13	6	117	24
Total	26	15	234	60

TABLE 7.—*Results of Intradermal Tests with Foods and Inhalant Group*

	Number of Persons Tested	Number of Reactors	Number of Tests	Number of Positive Tests
Lid type	16	16	879	304
Limbic type	37	36	1,634	517
Total	53	52	2,513	821

TABLE 8.—*Results of Ophthalmic Tests with Pollens of Trees, Grasses and Weeds*

	Number of Persons Tested	Number of Reactors	Number of Tests	Number of Positive Tests
Total	102	46	582	103

TABLE 9.—*Data on Conjunctival Smears Showing Eosinophils*

	Number of Smears Taken	Number Showing Eosinophils	Highest Count, Percentage
Lid type	18	17	96
Limbic type	49	21	78
Total	67	38	

TABLE 10.—*Data on Blood Smears Showing Eosinophils*

	Number of Smears Taken	Number Showing Eosinophils	Highest Count, Percentage
Lid type	15	10	22
Limbic type	22	10	18
Total	37	20	

Of the 102 persons on whom the ophthalmic test was performed with the dry pollen of the grasses, trees and weeds, 46 gave positive reactions. In a total of 582 ophthalmic tests, 103 positive reactions were obtained (table 8).

A total of 67 conjunctival smears were taken. Of this number, 38 showed a proportion of eosinophils greater than 4 per cent. These smears represented 18 cases of the lid type and 49 of the limbic type. The highest count was 96 per cent in the lid type and 78 per cent in the limbic type (table 9).

Blood smears were taken for 37 patients (table 10). Twenty showed a proportion of eosinophils over 4 per cent, the highest value being 22 per cent for those with the lid type of conjunctivitis and 18 per cent for those with the limbic type. Fifteen of the 37 patients had the lid type of the disease and 32 the limbic type.

COMMENT

We are here concerned primarily with evidence that this disease resembles in many ways other atopic states. The shock tissue is the ocular conjunctiva, while in hay fever it is the nasal mucous membrane; in chronic asthma, the bronchial mucous membrane, and in eczema and urticaria, the epidermis. As an atopic disease, vernal conjunctivitis is a form of hypersensitiveness subject to the influence of heredity. We have shown that heredity plays an important part in this disease and that the age factor corresponds to the age incidence of other atopic disorders. By laboratory tests, both intradermal and ophthalmic, we have shown a percentage of positive reactions comparable to those found in other allergic diseases. Vernal conjunctivitis in our series was shown to exist in the same family and also in members of families in which other atopic diseases were present. The similarity of the pathologic process and symptoms of this disease to those of other atopic states is evident.

The periodicity and seasonal recurrence of vernal conjunctivitis are definitely proved facts. Patients with vernal conjunctivitis, like many atopic subjects, have distress manifested mostly in the evening, just as does the asthmatic person and those affected with urticaria and eczema. The picture is much like that of pollen hay fever in which at first there are paroxysmal seasonal attacks, which gradually become complicated by seasonal asthma, become prolonged each year, continue after the pollinating season and finally become perennial without reference to any particular pollen.

Vernal conjunctivitis of the lid type resembles pollen hay fever in that there are seasonal attacks, each season adding to the pathologic involvement, with an aggravation of symptoms, until there develops a perennial pathologic condition with but seasonal symptoms. By this is meant that the simple follicular type develops into the cobblestone type, and the pathologic process may be recognized in the winter months with or without mucous discharge and with symptoms of itching only when the patient is exposed to heat or to some offending substance with which

the eyes may come in contact. The occasional symptoms during the winter are due to the persistency of the pathologic process and the complication by allergens other than those found during the seasonal attacks.

It has been definitely established that there is a specific local hypersensitiveness of the conjunctiva in tuberculosis and hay fever as demonstrated by the ophthalmic reaction—a specific sensitization without a constitutional reaction. In vernal conjunctivitis this sensitization has also been demonstrated as a result of an allergen coming into contact with the sensitized cells.

We do not regard this as a contact conjunctivitis. In the contact allergies the oil fraction of the antigen is of prime importance. Repeated patch tests, although not recorded in our results, have been performed on many of our patients and have been consistently negative. Further investigation with the patch tests along this line of endeavor is desirable. (This particular part of our research was suggested by Dr. Louis Tuft, chief of the allergy clinic, Temple University.)

In the process of our work other problems presented themselves to us, which, owing to insufficient time, could not be carried out. These problems consist of determining the effect of an air-conditioned room on patients with vernal conjunctivitis, including the effect of temperature and humidity, and the effect of the removal and introduction of various pollens, antigens and molds. (All patients who attended air-conditioned theaters reported prompt relief from ocular symptoms.)

The study of molds and fat-soluble excitants comprises a study in itself, and the effect of physical agents, such as light or photosensitivity, is another problem to be presented in the future.

SUMMARY

There is a definite family or personal history of allergy in patients with vernal conjunctivitis comparable with other forms of atopy.

There is a definite seasonal incidence.

The age group and sex incidence correspond to those found for other atopic states.

Laboratory studies reveal a large proportion of positive intradermal and ophthalmic reactions to the pollens and other antigens.

There is a definite increase in the eosinophils of the blood and a demonstration of these cells in the ocular discharge.

Epinephrine hydrochloride instilled in the eyes gives temporary relief from subjective symptoms.

There is a specific local hypersensitiveness of the conjunctiva without constitutional reaction.

The similarity between vernal conjunctivitis and other atopic diseases is striking.

AIDS IN THE FITTING OF CONTACT LENSES

HARRY EGGERS, M.D.
NEW YORK

The fitting of contact lenses can be expedited by a saving of time and trouble in the determination of the visual curve. The usual procedure requires measurement of the radius of curvature of the anterior corneal surface with the ophthalmometer and determination of the apparent axial refraction of the eye by neutralization of the patient's spectacle lens. Whenever astigmatism is present the radius of either one of the principal corneal meridians as well as the axial refraction of the eye in that same meridian must be measured. Application of these two measurements to either the graph or the scale supplied by the Zeiss Company will give the approximate required curve for the inner surface of the corneal segment of the contact lens. An afocal contact lens with this corneal radius is then placed over the eye. Finally, additional spherical lenses are placed before the eye in a trial frame until the proper visual correction is obtained. The final prescription will likely specify this corneal curve plus the additional spherical power, if any, that was added in the trial frame.

The procedure just described can be shortened by the elimination of the ophthalmometric determination and by the substitution of an instantaneous formula for the graph and table. Also, additional spherical power in a trial frame can be converted rapidly into an equivalent larger or smaller corneal radius in the contact lens.

Two formulas, one for myopic patients and the other for hypermetropic patients, were obtained in the following manner from the data secured by examining 152 patients for contact lenses. Two series of points, one for persons with myopia and the other for those with hypermetropia, were plotted on cross section paper. The abscissas were made to represent the total amounts of refractive power, in diopters, in the patients' spectacle lenses; the ordinates were made to indicate the corneal radiuses of the corresponding correcting contact lenses. The total amount of refractive power in a spectacle lens means the cylindric component added algebraically to the spherical component. It was assumed that the spectacle lenses were correct if they gave vision of 20/25 or better and if they were comfortable. An average locus of all of these points was drawn. For both groups of patients it was found to be a straight line. This made it easy to obtain an equation expressing the relation between the amount of refractive error and the required radius

of curvature for the corneal segment of the correcting contact lens. From analytic geometry it is known that a straight line is determined by two of its points and that the equation of a line passing through two given points P_1 (x_1, y_1) and P_2 (x_2, y_2) is:

$$\frac{y - y_1}{x - x_1} = \frac{y_1 - y_2}{x_1 - x_2}$$

By substituting the actual values of two of the points in the line in this equation in each case, two approximate empiric formulas were obtained.

For myopia, the following formula is applicable:

$$y = \frac{x + 30}{4}$$

In this formula y is the radius of the corneal portion of the contact lens, expressed in millimeters, and x is the total minus power, expressed in diopters, in the patient's spectacle lens (spherical moiety is added algebraically to the cylindric). This formula will give the radius of curvature for the corneal segment to within 0.3 mm. in almost every instance. This is surprisingly accurate when it is realized that many persons with myopia wear glasses which overcorrect to varying degrees.

An interesting and useful correlation was observed between additional spherical power as provided by lenses in a trial frame and the equivalent effect produced by changing the radius of curvature of the corneal portion of the contact lens. It was found that a spherical addition of 0.5 diopter produced the same effect as changing the radius of curvature by 0.1 mm. Instead of adding x diopters of plus or minus sphere in a trial frame in front of a contact lens, one can obtain the same visual effect by changing the radius of curvature by 0.2 x millimeters, adding this amount to the radius of curvature when the additional power is minus and subtracting it when the additional power is plus. For example, if a patient is wearing a contact lens with a corneal curve of 8.3 and it takes an additional minus sphere of 1.5 diopters power to improve the vision to 20/20, the required curve for the corneal part of the contact lens is 8.6. If a lens with this curvature is available in the trial case, it then can be inserted and the patient will see just what the final visual result will be. Likewise, if a plus 2.0 diopter additional sphere gives better or more comfortable vision to a person with hyperopia wearing a well fitting trial contact lens of 12.0/6.8 size, the final lens may be ordered as 12.0/6.4 to obtain the same visual result.

The empiric formula for quickly determining the corneal radius of a contact lens has already been given for myopia. The formula for hypermetropia is given only tentatively and with some hesitation, as it probably is not based on enough cases, only 41. Also, hyperopia is much less uniformly dealt with by oculists than myopia. Some ophthalmologists give full correction; others prescribe a minimum correction. Thus.

in hypermetropia the ciliary muscles do varying amounts of the work, and any formula, regardless of the number of cases on which it might be based, would be less exact than a formula for myopia. Therefore, the following formula should be regarded merely as a rough guide:

$$y = \frac{34 - x}{4}$$

In this formula y equals the radius of curvature, expressed in millimeters, of the corneal segment of the correcting contact lens; x equals the total plus power, expressed in diopters, in the patient's spectacle lens, the spherical and cylindric components having been added algebraically.

Similar plottings were made to discover any possible relation between the scleral curves of the correcting contact lenses and the amount of ametropia corrected by the spectacle lenses. No true correlation was found. Only a few generalities were observed. The scleral curves ranged in size from 11.4 to 13.2. Persons with myopia require only a slightly larger average curve than those with hypermetropia. Eighty per cent of those with myopia need a curve of 12.0 or larger, the most probable one being 12.6. For myopia of 5 diopters or more, a curve of 12.2 or larger probably will be needed. The majority of persons with hyperopia need a curve in the range between 11.8 and 12.5, with 12.0 as possibly the most likely curve for the scleral segment.

LOUIS ÉMILE JAVAL

1839-1907

A CENTENARY TRIBUTE

JAMES E. LEBENSOHN, M.D., PH.D.

CHICAGO

Javal had a genius for an infinite capacity for work. He was inspired in youth by the life and achievements of Benjamin Franklin; and the portrait of this guiding spirit ever after graced his study, and Franklin's admonition that "time is the stuff life is made of" became a motto, ever to be remembered. Javal himself was of the Franklin type and emulated his ideal in consistent industry and manifold interests; for Javal was not only an ophthalmologist but a legislator, journalist, educational leader, hygienist and social reformer.

When in 1806, at the suggestion of Napoleon, the Jews of France changed their oriental patronymics to the type of surname used by the rest of the French citizenry, Javal's grandfather inscribed his name Jacob on the register. This was miscopied by the clerk as Javal and became the family name thereafter. Leopold, Javal's father, was an associate of the celebrated financier Jacques Laffitte and for fifteen years was a deputy to the national assembly. In 1838 he married Augusta de Loemel, the cultivated daughter of a leading banker in Prague. Imbued with the idea of progressive education expounded in Rousseau's "Émile," she named her first child (the subject of this sketch), born on May 5, 1839, after the book and reared him by its tenets.

TRAINING

Émile Javal derived from his father the spirit of enterprise, from his mother a devotion to service and from both a keen intellect. Not for him the gay life of a rich man's son; his way was rather that of the practical idealist, zealous in work, simple in tastes, easy of access. A sincere friend, he was rewarded with the lasting friendship of many destined for high places. Among these were his classmates at the *lycée,* the brothers Carnot and Sully-Prudhomme, the poet, who later dedicated to Javal some of his charming verses.

From the Department of Ophthalmology, Northwestern University Medical School.

Javal inclined to a scientific career and could not be persuaded to follow his father's footsteps. He originally preferred medicine but, bowing to family opposition, compromised on mining. After his graduation at the age of 23, he spent a year in observing the principal mines of Europe and then accepted an engagement in the coal mines controlled by his family. Sundry innovations which he then proposed were rejected but later were introduced with success, a tribute to his insight.

Javal's interest in physiologic optics was aroused by several circumstances. His father had a convergent squint, for which Desmarre operated when Émile was 7 years of age. The result was a disfiguring divergent squint, which rendered the sympathetic family desolate. Javal's sister, Sophie, 15 years his junior, inherited the father's anomaly, a misfortune which later affected his brother's two children. Von Graefe, visiting Paris, was consulted by the father, and young Javal was profoundly impressed by the great personality. Javal himself had a minor ocular abnormality; one eye was blue, the other brown. Moreover, reading at night produced discomfort. A disciple of Donders dismissed his refractive error as "physiologic astigmatism," but Javal, while experimenting with a cylinder that he chanced to pick up, noted a definite improvement of vision. Perhaps there was a need for more refined methods for accurately determining astigmatism. The Brewster stereoscope, invented when Javal was 10, became one of his most vivid childhood memories. Perhaps it could be used to develop the faculty of binocular vision. These two problems immediately challenged his attention. His first studies, published in the *Presse scientifique des deux mondes,* received such encouraging attention that he abandoned mining, which he had practiced for a year, and enrolled in 1865 in the school of medicine of the University of Paris.

ACHIEVEMENT

Javal embarked on his new career with a display of amazing industry. Before he graduated he had improved the optometer and contributed sixteen articles on astigmatism and orthoptics to journals of ophthalmology. A linguist, able to speak French, English, German and Italian, and to read Spanish, Portuguese, Dutch and Latin, he sought to popularize in France the master works of Donders and Helmholtz. The 1864 English edition of Donders, translated with the aid of Debove, Rendu, Monoyer and Hunt, an American, appeared in 1866. At the suggestion of Gavarret, his professor in medical physics, Javal next proceeded, with the cooperation of Klein, to the translation of the work of Helmholtz, translating the last section from the manuscript, so that the French version and the completed German text were published

simultaneously in 1867. In this same year, Javal, then 28, married Maria Ellisen, of Frankfort. The following year, Javal graduated; his thesis, honored by a silver medal, was entitled "Du strabisme dans ses applications à la théorie de la vision." Javal then hurried to Berlin and imbibed the wisdom of ophthalmology from von Graefe, till his stay was interrupted by the Franco-Prussian War. He returned to France with his wife and 9 month old child and immediately volunteered as a medical officer for the service of his country.

After the war Javal became absorbed in civic issues, education and social reform. He followed his father's lead in politics and for twenty-four years represented his canton and for five years (1884-1889) was

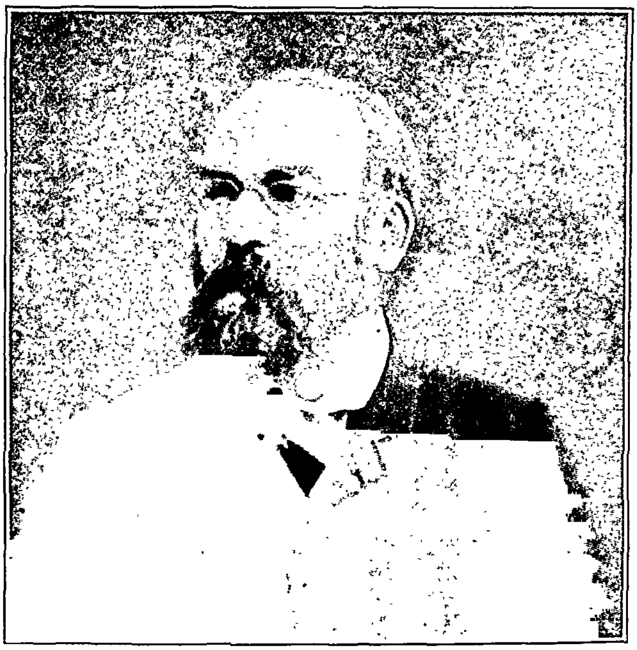

Fig. 1.—Louis Émile Javal. (From Ann. d'ocul., 1938, vol. 175.)

deputy from his department. Javal opposed the French construction of the Panama Canal and prophesied disaster for the project. Fearful of depopulation, he sponsored a law relieving families of seven from all direct taxes. Though the Javal law remained in force but a year, exemption privileges of similar pattern have been lately reconsidered. The influence of Javal's studies on the factors affecting population is reflected also in the novels of Zola. An ardent advocate of adult education, he was, with Camille Flammarion, among the charter members of an association for the popular diffusion of scientific knowledge and for many years was its president.

Javal's inability to secure an appointment to the department of ophthalmology of the University of Paris dampened for a time his scientific

enterprise. But in 1878 his old teacher, Gavarret, persuaded the faculty of general physiology of the Sorbonne to establish under its control a laboratory of ophthalmology and to name Javal its first director. The laboratory started as a single room with meager appointments. The famous library of Sichel, consisting of 8,000 precious volumes from the previous three centuries, was purchased from the heirs by Javal and presented to the institution. One of Javal's earliest co-workers was Schiötz, and in 1880 the two began work on the simplification of the ophthalmometer. Tscherning joined them in 1884. The laboratory soon achieved international recognition. Among those attached to its staff were Landolt, the elder Nordensen, Ericksen, Leroy, Sulzer and Bull, an American. Javal had justified his leadership, and in 1884, at the age of 45, he was elected a member of the Academy of Medicine, a signal honor for an ophthalmologist. Just then, at the height of his career, came the threatening symptoms of his eventual amaurosis; this but spurred him to give to the world in definitive form his knowledge of astigmatism, orthoptics, visual hygiene and blindness.

Javal's enthusiasm for ophthalmology centered in optics, and his practice was restricted mostly to refraction and squint. As he was of independent means, his office provided material for private study rather than a source of income. If a patient proved interesting, he would waive the fee and invite him to dinner. Once a month he visited the villages in his canton and gratuitously gave his services to the indigent, a tradition which Tscherning generously continued, but otherwise Javal never conducted a clinic. Uninterested in surgery, he referred all operative interventions to his colleagues.

Every phase of practical optics excited Javal's interest. A vexatious lawsuit followed a report in which he deflated the advertised claims of some lenses made of baryta glass. An enlightened court was fortunately for scientific freedom. Javal's earnest support hastened the general adoption of the dioptric enumeration proposed by Monoyer. The translucent test chart used with a mirror was his device. It was also his suggestion that plus and minus lenses be distinguished in trial cases by silver and gold rims, respectively, silver for the silver hair of presbyopia.

After Young made his discovery of astigmatism, the use of the optometer to test astigmatism continued till Javal constructed the most perfect form of this instrument. In Javal's optometer (fig. 2), presented in 1865, a convex lens was placed in the ocular and the radius figure moved away until but one line was most distinct. Concave cylinders were then introduced at right angles to this axis till all parts of the figure became about equally clear. Javal introduced a numbering of cylinder axes that has since become standard, that is, from left to right,

0 to 180. The monocular findings were finally checked binocularly. With this optometer he secured the first successful correction of his own astigmatism and demonstrated also the astigmatism that occurs after extraction of cataract.

The elder John Green, then in Snellen's clinic, realized that consciousness of the close proximity of the object prevented the accommodation from completely relaxing. He adapted Javal's method for use at 20 feet (6 meters), thus initiating the present procedure, by substituting for the optometer diagram a large chart with sixty radiating lines at intervals of 10 degrees.[1] The space-saving "astigmatic fan" of Snellen, published in 1875, is but the upper half of Green's chart. Holth [2] incorrectly credited Javal with the invention of the radius figure, but Burow demonstrated an optometer with a similar diagram in 1863. Burow borrowed the stelliform figure from Donders (1860), and Donders from Helmholtz. The familiar clock dial, however, was Javal's own innovation.

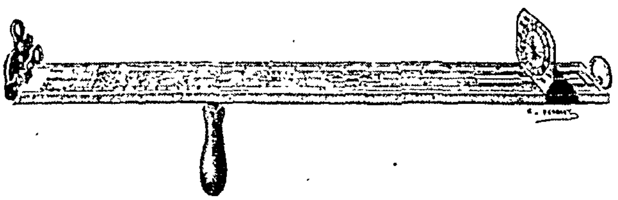

Fig. 2.—Javal's optometer.

The clinical ophthalmometer of Javal and Schiötz was hailed in its day as the greatest contribution to ophthalmology since the ophthalmoscope. A preliminary advance was made by Coccius, who introduced in the Helmholtz instrument the birefringent crystal (1872). Javal's ideal of further simplicity and precision was realized after four years of patient labor. With his perfected instrument he completed more ophthalmometric measurements in one day than had been accomplished by all observers with the Helmholtz apparatus in the previous twenty-five years.

The earliest reports of astigmatism, in which the observers cited their own defects, were considered curiosities. Even in 1854, Isaac Hays, of Philadelphia, published as noteworthy reports of 3 cases of

1. Green, J.: On the Detection and Measurement of Astigmatism, Am. J. M. Sc. **53**:117, 1867.

2. Holth, S.: The First Radius-Figure for Subjective Astigmometry, Brit. J. Ophth. **20**:415, 1936.

astigmatism in which correction was obtained with cylinders. The prevalence of astigmatism, first suspected by Goulier (1852), was firmly established by Donders (1862). Donders regarded the common errors of low degree as within normal tolerance, but Javal insisted that the relief of visual fatigue required even their meticulous correction. By continually emphasizing that the normal eye does not readily tire, he promoted better refraction and overcame the previous hesitancy in prescribing cylinders. Since a dilated pupil exaggerates the defects of astigmatism, he advised testing in reduced illumination. Not having the advantages of a cross cylinder, he resolved doubts as to the exact axis by tilting the patient's head alternately to the right and left; if the vision seemed improved with the head tilted to the right shoulder, a shift of the cylinder to the right was indicated.

ORTHOPTICS

Javal's younger sister, who is still living in Paris, has the distinction of being the first person in the world to receive orthoptic training. Sichel, who saw her at age 2 years, prescribed alternate occlusion. When she was 10, Javal started his experimentation, Giraud-Teulon having tried prisms without avail, but after six years he was only partially successful. When the sister was 16 von Graefe performed tenotomy, which likewise failed to correct the condition fully, whereupon Javal resumed his methods and achieved a perfect cure, which has persisted to this day.

Javal revived Buffon's view that squint was an anomaly of binocular vision and rejected the current idea that the ocular muscles were primarily at fault. He began with the hope that the orthoptic attack would eliminate any need of surgical intervention, but finally concluded that a better management of squint was assured by a partnership of operative aid and functional education. With the courage of his convictions, he had de Wecker perform tenotomy on his 16 month old nephew. The child then wore a shield constantly on one eye or the other for the next seven years. Stereoscopic exercises were started at the age of 9 and continued for three and one-half years, with the ultimate reward of a normal appearance and stereoscopic binocular vision.

In Javal's system, explained in his "Manuel du strabisme" (1896), the successive objectives were to overcome suppression, to train fusion and to establish the correct relation between accommodation and convergence. For children under 6, Javal restricted his treatment to accurate refraction and to promote normal projection, he kept one eye or the other constantly occluded. The use of the shield was not entirely abandoned until after adequate binocular training. Between the ages of 8

and 10, when the child's attention and obedience were at their best, was his preferred period for stereoscopic exercise. After he had taught binocular vision with his hinged stereoscope, fusion amplitude was trained by narrowing the angle of the mirrors. Shifting then to the lenticular stereoscope, he began with large letters and words, gradually decreasing to those of smaller size. The lenticular stereoscope, consisting of 10 diopter lenses, was capable of three major variations, in the distance between the lenses, in the distance between the centers of the cards and in the distance between the card and the lenses, so that with proper manipulation accommodation could be stimulated without convergence and vice versa. Exercises were provided for home training with the simplified stereoscope popularized by Oliver Wendell Holmes. As a further help for primary convergence excess, bifocal lenses were advised. *The final training of the patient was in bar reading of ordinary* type.

Javal's procedure was derived from an investigation of 440 private patients. Not all required prolonged attention; several with secondary divergent squint after operation were cured in from four to ten days. The results obtained with certain patients, for instance, a niece, were not considered worth the time and effort required. Though Javal developed orthoptic training, Hirschberg [3] discovered that to Dubois-Réymond belongs the first suggestion of using the stereoscope in this field (1852). Hirschberg also took exception to Javal's introduction of the term amblyopia ex anopsia, for which he would substitute as etymologically more correct amblyopia ex ablepsia.

VISUAL HYGIENE

Javal's discovery of the character of ocular movements in reading in 1879 was a by-product of his research in orthoptics. To detect when eyes were used binocularly or alternately, he asked his student Lamare to watch their action behind a glass plate on which horizontal strips of print had been pasted. The saccadic movements then revealed laid the basis of an objective analysis of reading ability that has since culminated in instruments for recording ocular movements and for training efficient reading habits.

In 1885 Javal proposed an original method for teaching reading and writing simultaneously and, with the collaboration of two of his daughters, prepared illustrative textbooks (fig. 3). To accelerate learning, he introduced letters in their order of frequency. With "a-b-c-d" no phrase

3. Hirschberg, J., in von Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 2, Berlin, Julius Springer, 1918, vol. 15, pt. 2, p. 536.

formation is possible, but with "r-a-l-i" the child could write "Lili a ri," and "Lila rira" in the third lesson. By the thirteenth lesson, he was reading and writing stories even though he knew but half the alphabet.

MÉTHODE JAVAL

La Lecture

Enseignée par l'Ecriture

PAR

LE DOCTEUR JAVAL

Ingénieur des Mines,
Membre de l'Académie de Médecine,
Vice-Président du Cercle parisien de la Ligue de l'Enseignement,
Lauréat de l'Institut (Prix Monthyon)
Officier de la Légion d'Honneur, Officier de l'Instruction Publique.

PRÉFACE

DE

M. I. CARRÉ

Inspecteur général honoraire de l'Enseignement primaire,
Officier de la Légion d'honneur, Officier de l'Instruction publique.

Premier Livret

Depuis Novembre 1893, l'Écriture droite est acceptée pour l'examen du certificat d'études et du brevet de capacité.
(Décision ministérielle.)

PARIS
LIBRAIRIE D'ÉDUCATION NATIONALE
Publications Alcide Picard
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(Droits réservés)

Fig. 3.—Javal's reading primer.

Javal emphasized hygienic school construction, proper posture and efficient writing habits. With many educators of that period, he favored vertical penmanship, a beautiful example of which is the hand of Thomas A. Edison. His studies of the effect of variations of light, paper and print on the ease and speed of reading foreshadowed the modern interest in the subject. Considering the problems of typographic

legibility, he sought with the aid of his assistant Sulzer to determine the most readable type in feeble light. In his analysis of typography he surveyed all writing symbols from cuneiform inscriptions to musical notation. He suggested that the change of pens from quill to steel caused the handwriting to alter from Gothic to angular. Ever motivated by humanitarian ideals, he stressed that to facilitate reading and writing is to accelerate communication among men.

BLINDNESS

Unfortunately for Javal, the modern operations for glaucoma simplex, as exemplified by sclerectomy, trephining, cyclodialysis and iridencleisis, were introduced after his eyesight had been destroyed by the disease and ineffective intervention. In 1883 the first premonitory attack affected the right eye, and the election campaign of 1885 provoked a severe exacerbation. In August of that year the left eye also began seeing halos and was kept for the next fifteen years constantly under the influence of pilocarpine. Noyes, who chanced to examine Javel in 1885, observed a deep excavation of the right disk and advised operation. Sclerotomy performed on November 10 and on December 3 of that year failed to control the tension, and iridectomy performed on December 11 proved disastrous; the vision, which had been 20/20, was lost completely.

After finishing his term as deputy in 1889, Javal devoted himself wholly to scientific interests. In 1890, at the banquet of the International Congress of Ophthalmology at Berlin, where he was seated with Jonathan Hutchinson and other distinguished guests, his fervent response to Hirschberg's address of welcome ended in a spell of obscuration. Hirschberg then noted in the left eye an excavated disk with a spontaneous arterial pulse. Javal, however, continuing with his work, in 1891 edited his "Mémoires d'ophtalmométrie" and in 1896 released his "Manuel du strabisme," which epitomized thirty-three years of experience and observation.

In 1897 the excitement of the Dreyfus trial at Rennes precipitated an attack in his only seeing eye that left him blind for some hours. Now keenly aware of his prospective doom, he prepared his notes for easy accessibility so that whatever happened he could carry on his appointed tasks. The light sense progressively diminished, until finally red looked like black. In February 1900 he journeyed to Birmingham, where Priestley Smith performed a futile iridectomy, using cocaine hydrochloride anesthesia; in five months Javal was sightless. Later in Paris the cervical portion of the sympathetic trunk was denuded, but without benefit. Command of the laboratory was transferred to the able leadership of Tscherning, and Javal became honorary director.

Stark blind at 62, Javal resolved to imitate those brave souls like Euler, Huber, Milton and Fawcett, who had not been deterred by a like fate from magnificent achievement in such diverse fields as physical and natural science, literature and public affairs. In 1901 he invented a writing rack and published in vivid detail his auto-observations on glaucoma. In 1903 came his most widely circulated volume, "Entre aveugles," translated into German by Turkheim and into Esperanto by his daughter-in-law and available to English readers in two versions, "The Blind Man's World," by Ernest Thompson, of England, and "On Becoming Blind," by Miss Carroll Edson, of the United States. To

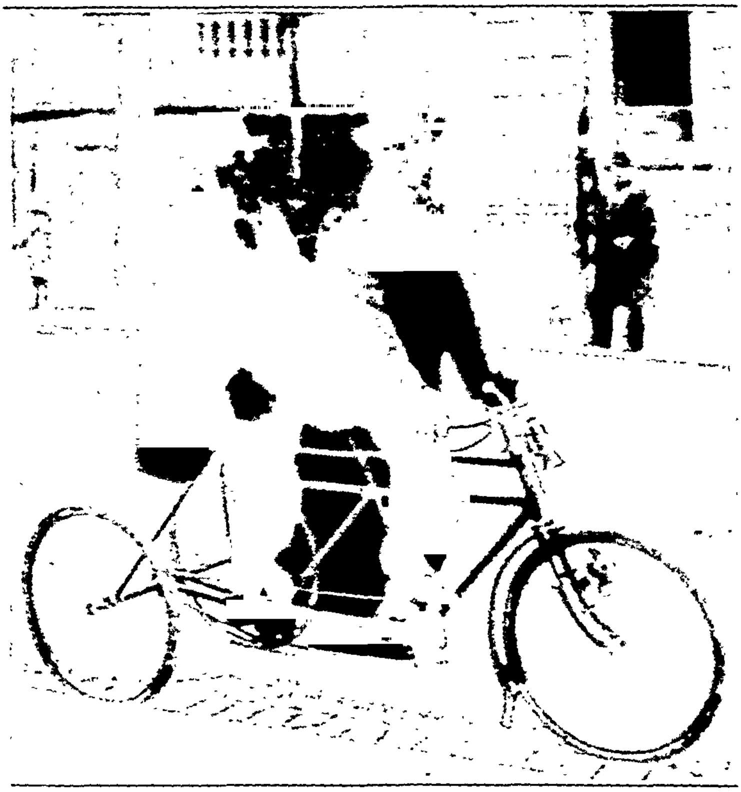

Fig. 4.—The blind Javal taking his exercise on tandem bicycle.

Javal, dependence was the chief misery of blindness, and he aspired in his book to help the blind to help themselves. He advised paid help, when possible, as preferable to voluntary attendance. He encouraged physical as well as intellectual activity, and by means of a tandem bicycle he continued regularly his favorite exercise (fig. 4).

About this time Javal became a passionate Esperanto enthusiast and financially supported a central bureau in Paris for its advancement. He pleaded for the general adoption of this auxiliary language, which would be of especial advantage to the blind, since it would permit an international use of Braille publications. To his friend Zamenhof, a Warsaw oculist who invented the language, he made various acceptable

suggestions to render Esperanto even simpler. To Zamenhof was dedicated his following work, the comprehensive "Physiologie de la lecture et de l'écriture" the first edition of which, published in 1905, was exhausted in three months.

Javal was working on the second edition of this text when illness intervened. The invitation of the Ophthalmological Society of the United Kingdom to give the Bowman Lecture for 1907 had to be declined. Death came on Jan. 20, 1907, from gastric cancer after five weeks of intense suffering. Javal had written Priestley Smith that he would will the eye that had been operated on to him, so that he should not consider the operation a total loss. The promise was kept; after

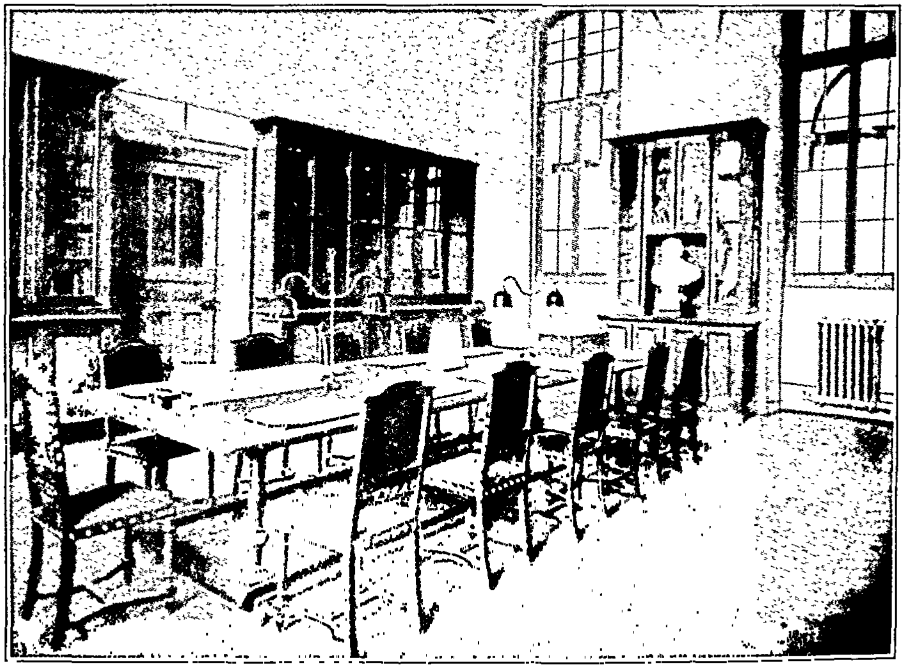

Fig. 5.—Bibliothèque Javal, at the Clinique ophtalmologique de l'hôtel Dieu.

Javal died, Tscherning enucleated the eye and dispatched the visual organ in preservative to Birmingham. In accordance with Javal's behest, the body was cremated at Père Lachaise with private obsequies. But throughout the civilized world scholarly journals and organizations paid homage to the passing of a personage.

Javal had five children (two sons and three daughters) and twelve grandchildren. Though he was reared in traditional Judaism and never deviated from his formal allegiance, he was devoted to the ideal of universal brotherhood and insisted that the spiritual and ethical education of his children be free from all dogma. His son, Jean, a civil engineer, who succeeded to his place in the national assembly, lost his life in the

World War. His other son, Dr. Adolphe Javal, studied under Widal and with him investigated the purification of water by chlorine. He recently published his experience with military sanitation during the war ("La grande pagaïe," 1914-1918). He never entered private practice and since demobilization has been occupied chiefly with literature and economics.

After Tscherning was called to assume the chair of ophthalmology at the University of Copenhagen in 1909, the laboratory of ophthalmology at the Sorbonne was closed and the space reassigned to the department of physiology. The famous library of Sichel and Javal remained conserved by Professor Dastré till 1914, when at the instance of Javal's widow the library was completed and transported to suitable quarters in the Clinique ophtalmologique de l'hôtel Dieu. A splendid bust of Javal by Verlet was presented to grace the reading room. On Feb. 18, 1914, the bibliothèque Javal was dedicated, a fitting memorial to one of the great scholars of ophthalmology (fig. 5).

Javal's children, Dr. Adolphe Javal and Mlle. Mathilde Javal, gave me access to information, material and portraits not otherwise available.

PSYCHOLOGIC CONSIDERATIONS IN THE STUDY OF BINOCULAR FUNCTION

EMANUEL KRIMSKY, M.D.
BROOKLYN

The study of binocular function requires a psychologic as well as a clinical understanding of individual responses more than other branches of ophthalmologic investigation. Physicians trained mainly in established clinical methods may neglect this psychologic approach to the study of binocular function and the need for binocular training.

As a prerequisite to this approach, one should know (1) what constitutes an average normal binocular response on a binocular instrument, such as a synoptophore or a calibrated stereoscope; (2) how such readings may be correlated with established clinical methods of examination; (3) in what different ways binocular imbalance may become manifest, and (4) to what extent abnormal responses represent psychologic or functional impairments.

The psychologic elements are interrelated with certain physiologic responses. These are represented by the muscular movements concerned in the changing fixation of the two eyes that follow two targets which move toward or away from each other, as in the stereoscope, namely, the disjunctive movements of the eyes. The psychologic aspect concerns itself with the ability of two eyes, made to work independently by the interposition of a septum (as in a stereoscope), to fuse the corresponding images into a single image.

A clinical study concerns itself with qualitative reactions and also with an actual quantitative measurement of the disjunctive movements of the eyes. It must also consider coexisting psychic fusional registrations.

It is difficult to disassociate the intricate psychologic, innervational, structural, developmental, accommodative and even chemical changes which may take part in the infrequent spontaneous correction of a binocular imbalance. It also cannot be explained satisfactorily how orthoptic training improves binocular status. One can point only to end results, and then speculate vaguely on underlying processes.

Were it possible to think of muscles as elastic bands it would be simple to explain such altered clinical phenomena. Luciani and other physiologists have credited muscle tissue with only slight elastic properties. More recently, certain physiologists, notably Hill [1] (1926) and

Read before the New York Society for Clinical Ophthalmology, Dec. 5, 1938.

1. Hill, A. V.: The Viscous Elastic Properties of Smooth Muscle, Proc. Roy. Soc., London, s.B **100**:108, 1926.

Levin and Wyman[2] (1927), have suggested that the mechanics of muscular contractions are governed by viscosity as well as by elasticity. In a mathematical and experimental treatment of the subject they showed that the facts can be accounted for on the assumption that the muscle is a complex system consisting of a free elastic part, comparable to an undamped spring, and a viscous elastic part, comparable to a highly damped spring. However interesting such isolated experiments may be, they clarify in but a small way the complex phenomena participating in ocular muscle response due to a combination of positive and negative forces subject to peripheral and central nervous control.

Let a muscle response, such as abduction, be considered not simply as a contraction of the lateral rectus muscle but also as a stretching of the medial rectus muscle. (Stretch limit is a physiologic term applied to muscles.) With such an inclusive conception, one strives, say in a case of convergent squint, to increase the elastic abduction response; in other words, figuratively to stretch the internal rectus muscle as if it were an elastic band.

Resiliency or recoil in an elastic body, such as a spring, is governed by the degree to which it is stretched. When a spring has been stretched by a load, it recovers its original length and elasticity after the removal of the load, provided the stress by the load does not exceed a certain threshold value. This value is called the elastic limit. With stresses beyond the elastic limit, the power to recover the original durable form or size, namely, the elastic property, becomes less. The recovery point of the stretched or lengthened substance approaches more and more the elastic limit. In the case of binocular response, the elastic limit would correspond to the "breaking point," or limit of muscle duction, and the "recovery point" would represent the status quo, or position of rest from which such stretching or ductions could again be resumed. In a case of squint it is important not only to raise the duction, or "breaking point," of the excessively strong muscles but to diminish their relative elasticity, namely, to raise the "recovery point" to approximate the "breaking point."

A binocular study on a synoptophore or a calibrated stereoscope[3] should include:

1. Measurement of the angle of squint
    (*a*) Objectively, by observing the corneal reflections of lighted targets brought in line with the angle of squint

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2. Levin, A., and Wyman, J.: The Viscous Elastic Properties of Muscle, Proc. Roy. Soc., London, s.B **101**:218, 1927.

3. Krimsky, E.: The Stereoscope in Theory and Practice; Also a New Precision Type Stereoscope, Brit. J. Ophth. **21**:161 (April) 1937; Some Newer Developments in Precision Type Stereoscopes, Arch. Ophth. **19**:394 (March) 1938; Descriptive Atlas of Orthoptic Slides, Tr. Am. Acad. Ophth., 1938, to be published.

(b) Subjectively, by observing whether the patient has: True projection or target correspondence at the angle of squint
False projection (abnormal retinal correspondence)

2. Measurement of stereoscopic esophoria, exophoria or vertical phoria for infinity equivalent or selective accommodational ranges
3. Measurement of fusion amplitude for far and for near vision
   (a) Abduction range—breaking point and recovery point
   (b) Adduction range—breaking point and recovery point
4. Measurement of vertical ductions
5. Determination as to whether stereopsis or third degree fusion exists and to what degree
6. Determination as to whether there is macular or paramacular suppression

This will enable one to classify a case of binocular imbalance as to: (1) convergence excess (esophoria or esotropia for near vision), (2) convergence insufficiency (exophoria or exotropia for near vision), (3) divergence excess (exophoria or exotropia for far vision) and (4) divergence insufficiency (esophoria or esotropia for far vision). It will also enable one to adjust the binocular training instrument to such particular type of dysfunction.

The principles governing the operation of a binocular instrument conform to simple and well recognized ophthalmologic methods.

From a psychologic standpoint, the following problems are considered: (1) the meaning of fusion; (2) duction readings in relation to psychologic influences; (3) the meaning of stereoscopic pictures; (4) the requisites of a binocular instrument, or a critical analysis of methods used in binocular study and training; (5) what to expect from orthoptic training; (6) the role of technician or orthoptist, and (7) orthoptic training as a problem for the ophthalmologist, or a comprehensive plan for closer cooperation between clinician and psychologist.

THE MEANING OF FUSION

The subject of fusion will, for the sake of brevity, be dismissed with but a definition of the term.

Fusion consists in the blending of two like or somewhat dissimilar (stereoscopic) pictures into a single image. It also indicates the ability of the eyes to superimpose two entirely dissimilar images. The mere act of fusion does not, in itself, necessarily indicate that the binocular status is normal. It merely shows that there has been a psychologic blending of two images. A quantitative study of fusion consists in the measurement of phoria, the fusion amplitude, the ability to fuse stereoscopic pictures, etc. It is only after these and other findings are recorded accurately that one may evaluate the fusion status.

DUCTION READINGS IN RELATION TO PSYCHOLOGIC INFLUENCES

There are numerous psychologic elements which no doubt play an appreciable role in varying the amount of fusional or duction response. Some of them are considered here.

(*a*) Worry, time of day and illness lead to a state of fatigue. A lengthy examination also is conducive to fatigue and to lack of attention. For this reason it is sometimes wise to make recommendations on the basis of repeated examinations rather than on a single study, no matter what method one employs.

(*b*) Concentration is an important factor in modifying duction readings. Psychologic influences appear to exert a much greater effect over disjunctive movements, such as adduction and abduction, than over conjugate movements. Even in such a simple clinical test as measuring the convergence near point, one must seek the patient's willing cooperation. The psychologic approach of the examiner is always a factor which may alter the readings. Lack of concentration may further be contributed by such elements as fear, noise or lack of privacy.

(*c*) The method of operating a binocular instrument, such as speed of excursion of targets, whether it is operated by the examiner or by the patient, also registers their influences. Furthermore, the types of pictures used in the carriers are of decisive value in winning the child's cooperation and will be dealt with separately.

THE MEANING OF STEREOSCOPIC PICTURES

The ophthalmologist is likely to be a little bewildered with stereoscopic pictures. They often appear meaningless and cannot be correlated with older clinical methods of study. Moreover, they look too complicated for the child, who can profit best from binocular study and training.

From the clinical standpoint one should, first, distinguish fusion from nonfusion slides; second, distinguish simple from difficult fusion slides; third, analyze third dimensional or stereoscopic pictures, and fourth, determine the retinal angles which targets subtend.

From a psychologic standpoint, my objections to many stereoscopic pictures are that they contain too much detail matter, have no major point of interest, are not sufficiently appealing to the young child, i. e., do not have enough human interest, and contain too much descriptive matter.

Even many of the pictures in children's picture books seemed interesting on the surface but were not simple enough for the younger child. A Walt Disney picture would prove uninteresting if it had too many unnecessary lines to distract the attention from a central focus of interest, such as a tree, a house or a path. I found that photographs of

simple toys, or colored drawings with the fewest lines would hold the child's attention. Pleasing pictures simplify the study of binocular function in the child and because of their psychologic appeal will often yield greater duction readings than poorer pictures. As in walking, there is also a psychologic element to ductions; for pleasing pictures serve the same purpose as nice trees and pretty scenery. In one instance the person instinctively makes more effort to increase his duction range; in the other, he enjoys his walk and proceeds farther with less effort.

THE REQUISITES OF A BINOCULAR INSTRUMENT

In order for a binocular instrument to have diagnostic value, it must be a well constructed unit and be so calibrated as to yield quantitative readings of the binocular status of the eyes in its various aspects. Only by careful, and occasionally by repeated, studies can one determine a deficiency in binocular function. Furthermore, without such measured readings of the binocular status one cannot have an intelligent basis for recommending binocular training. This must be adapted to the particular type of disturbance which the patient presents; namely, a binocular instrument must also possess diagnostic merits.

Instruments for orthoptic training present either a rotary or a horizontal type of motion. Conditions amenable to binocular training involve either the medial or the lateral rectus muscle. The problem is to turn the eyes out when they turn in, and in when they turn out. With a binocular instrument the aim is first to induce fusion and then to increase the duction range. Horizontal separation or approximation of targets carries out such a physiologic or psychologic function. I cannot conceive of rotary movements which increase the motility of the eyes in the cardinal directions to adduct or abduct them, as the case may require. Less ocular motility is required with horizontal excursions than with rotary movements. Slow, graded stretching is far more efficacious than a series of rapid rotary motions. The primary aim is to increase disjunctive duction range; rotary movements merely make one eye follow the other without necessarily affecting such duction.

Every binocular instrument presents certain psychologic features which appeal to the doctor or to the patient.

A flasher, for example, may be regarded as a psychologic device. It has some clinical merit in that it often does awaken a suppressing eye to function with its fellow eye. An elaborate automatic flashing unit has no appreciable therapeutic advantages over a simple hand-operated device.

A revolving, or a rotary, device for targets would be a psychologic feature the merits of which would seem to lie more in fancy based on the idea of motion of the eyes rather than on an intricate clinical or theoretic

analysis of the actual benefits of such rotary motion. I have seen no reason to incorporate this feature in a binocular instrument for the correction of squint.

Automatic motion of targets fits in with the present day psychologic concept that comfort and conservation of energy are things to be sought for. I have avoided automatic motion just because it renders such ocular training passive and is therefore conducive to laziness.

A calibrated stereoscope or synoptophore, which the patient can operate, with smooth and mechanical horizontal excursions of targets is the most logical device for binocular training. Such self operation would correspond physiologically to the voluntary movements of the eyes and psychologically would stimulate an active interest in improving binocular function. Normally the eyes do not move steadily from one fixation point to another but in rapid jerky fashion. After each rapid jerk there is a fixation pause, during which they remain relatively at rest. It would seem logical, therefore, that enforced exercises of the eyes should be based on such interrupted ocular movements, and with a hand-operated instrument they could be better controlled to correspond to the physiologic and psychologic peculiarities of the subject's eyes. In piano playing there is also an intimate association between the brain, the eyes and the fingers; in other words, a psychophysical parallelism.

WHAT TO EXPECT FROM ORTHOPTIC TRAINING

The most unfavorable aspect of orthoptics is that it has been unfairly exploited as a cure for cross eyes, without critical analysis of the binocular status of the eyes. Even the word orthoptics is faulty in that it creates suspicion in those who prefer to think primarily of binocular status rather than of binocular training. That term would imply that straightening of the eyes is the primary aim. As a matter of fact, what one strives for in orthoptics is, first, a functional result and, second, a cosmetic result. I object to the word orthoptics because it suggests impressive methods that will put the eyes in motion without critical reflection as to the fundamental psychologic or physiologic basis for such ocular calisthenics. I would substitute the term controlled binocular training to indicate that responses must be measured in order to have clinical value, whereas mere exercises have suggestive or autosuggestive value.

THE ROLE OF THE TECHNICIAN OR ORTHOPTIST

Orthoptics has created in the mind of the ophthalmologist the fixed idea that it cannot be practiced satisfactorily without the aid of a trained technician. I do not concur in this idea. My plan has been to examine the child with a major instrument in the office and then to explain to the intelligent mother or relative in simple terms what is being attempted in

order to correct a binocular dysfunction, and thereby to gain her cooperation as an intermediary in observing the child's training on a junior calibrated instrument at home under my periodic supervision, provided the eyes show true retinal correspondence. Children showing abnormal retinal correspondence are not suited to home training until such correction has been made in office or clinic. Fortunately, such patients are less common. The child is taught to keep records of progress and, if possible, to operate the instrument himself rather than to have the mother manipulate the targets.

The orthoptist may prove of value as a psychologic assistant to the ophthalmologist in certain selected cases. She may advise the mother as to teaching the child to concentrate on targets, selecting those pictures most suitable for the individual child, training the child to play an active rather than a passive role in stimulating increased ductions and keeping records of progress; she may also suggest to the ophthalmologist the psychologic aspects of the child's case, namely, the intelligence quotient, the child's determination to make headway and ways to improve the psychologic approach. It is far more important for the ophthalmologist to realize the problems of the psychologist than for the latter to study the clinical aspects.

ORTHOPTIC TRAINING AS A PROBLEM FOR THE OPHTHALMOLOGIST

The office or the clinic is not the most practical setting for orthoptic training. Binocular study and binocular training involve the expenditure of valuable time, which interferes with office routine. The use of a binocular training instrument in the office or clinic usually entails the hiring of an assistant to relieve the ophthalmologist. If training is to be recommended at all, it should be done daily rather than once or twice a week, as in the office or clinic. Even though binocular training is definitely helpful in selected cases, the length or duration of such treatment cannot be foretold and must be based on individual responses. And, finally, office or even clinic orthoptics is an expensive process.

The home is the ideal setting both for comfort and for concentration. Most clinics are not conducive to quiet and privacy, and even when such conditions are passable, the patient can return only once or twice a week. If exercises are to be continued for months, this may prove a drawback. Daily exercises at home would probably shorten the duration of such training.

What is one to do with the patient who cannot afford to rent a desirable instrument? I believe that the school system should set aside special periods during which the teacher, usually the kindergarten teacher, may train and interest the child in the operation of a binocular

instrument. I believe that the teacher as an educator thoroughly acquainted with the child as a child can adopt a sympathetic and patient approach which is impossible in the office or clinic. These children should be seen by the ophthalmologist at regular intervals to decide whether training should be continued or stopped or progressively increased. He may cooperate with the teacher through simple written instructions, without her having to be burdened with the clinical study of binocular function. The teacher may thus serve as the natural complement to the ophthalmologist in the psychologic training of binocular dysfunction. There are cardiac classes for school children. These children are regularly observed by physicians interested in that field. It does not necessarily follow that the children's hearts become normal, but they are being watched by competent men in their respective fields. Such allied fields as otolaryngology have found their entrance into the school system through audiometric testing, lip reading and other matters which are of psychologic as well as of medical interest.

I can conceive of the ophthalmologist working hand in hand with the educator, rather than being just a trained refractionist in the school system, as in the present setup.

CONCLUSIONS

1. A clinical study of binocular function should be a prerequisite to a psychologic approach to the problem. This should include a careful qualitative and quantitative instrumental study of the binocular status of the eyes.

2. The principles underlying the operation of a binocular instrument are simple and conform to recognized ophthalmologic theory and practice.

3. Duction readings are subject to psychologic influences, such as lack of concentration, will, nature of targets and environment.

4. The physician who has not mastered the diagnosis and measurement of binocular function is in no position to recommend binocular training.

5. A binocular instrument should provide a balance between psychologic and scientific features. It must provide a satisfactory means for measuring the binocular status of the eyes, otherwise it is unsatisfactory for binocular training.

6. The home would seem to offer a satisfactory environment for controlled binocular training in cases in which eyes show true retinal correspondence.

7. When binocular training is economically prohibitive, daily training should be provided in schools rather than in clinics, with periodic check-ups by a trained ophthalmologist.

8. The role of the orthoptist shall be that of psychologist or educator; the role of the ophthalmologist, that of clinician. Satisfactory binocular training may be supervised by the teacher without an intimate knowledge of binocular function.

DISCUSSION

Dr. LeGrand H. Hardy: Dr. Krimsky has done a great deal of splendid work, particularly in improving the stereoscope, but he has greatly and unjustifiably oversimplified the problem in order to present his point of view. The mechanics and physiology of squint are much more complex than he assumes.

I do not think that one is justified in treating fusion as the simple, although vaguely defined, process postulated by Dr. Krimsky. He has mentioned only one component of the process of fusion. This may be characterized as the cerebral component. Others, possibly as important, are the factors involved in conduction and in the formation of retinal images and, finally, the extraocular factors.

The conduction component offers an anatomic basis for a weak or absent fusion ability. It relates to the degree of decussation at the chiasm. Electroencephalograms may furnish important data for evaluating this factor. The factor concerned in the formation of retinal images involves differences in the sizes of the images and induced size effects, which likewise may have an important bearing on the problem of squint. Finally, the extraocular factors, the proprioceptors of the extraocular muscles, may have an important bearing both on the conception of the squinting process and on the surgical treatment.

Nor are the physiologic factors of stereopsis well understood. Most tests are based on pure disparity of imagery, which produces the illusion of depth. Many other factors enter into this sensation. I have mentioned ten ways of producing the illusion of depth in flat pictures. Few stereoscopic tests are based on actual depth.

Dr. Krimsky's discussion of the importance of the psychologic factors entering into the diagnostic and therapeutic methods used for squinters is excellent. Few ophthalmologists have the temperament, imagination or training for such work.

I disagree most emphatically with Dr. Krimsky's statement that the best place for training is under the supervision of a parent at home. If one limits the discussion to persons with phorias or tropias and well established normal retinal correspondence, this procedure has definite merits, but for 60 per cent or more of persons with tropias and anomalous retinal correspondence Dr. Krimsky's proposed procedure is extremely dangerous and is bound to lead to incalculable harm. No such patient should be given any orthoptic treatment unless it is under the direct supervision of an expert technician; otherwise all efforts will serve only to confirm rather than cure the squint and will make the complications more severe and difficult for the surgeon who finally has to operate. This particular misapplication of orthoptics has been the outstanding stumbling block and has caused the greater part of the discouragement, disappointment and disbelief in the method.

Clinical Notes

A COMBINATION OF THE SNELLEN AND THE LANDOLT TEST TYPES

David D. Waugh, M.D., Brooklyn

Since Snellen first presented his test types in 1862, numerous attempts have been made to improve them. His figures subtended an angle of 5 minutes, each element subtending an angle of 1 minute. Oblong letters, 5 minutes in height and 4 in breadth, have been suggested. Easily recognized confusion and difficult letters have been used. Lines, dots and concentric circles have had their trial. The recognition of the capital E has been used, especially for illiterates. In order to exclude the large psychologic factor, Landolt devised the broken circle as a measure of two point separability. This has subsequently been accepted as the international standard for tests of visual acuity. Broken squares of different sizes have also been used.

Time and experience have left only two types for routine office and clinic practice, the Snellen figures and Arabic numerals of a similar size.

A combination of the Snellen and Landolt test types.

It was felt that a combination of the popular Snellen types with the Landolt broken circle would be the next step in the development of the perfect test type. Investigation of the English alphabet revealed the fact that there are ten letters the base of which is a small round circle. For the visual acuity chart, this base is a circle subtending a visual angle of 3 minutes, having a central aperture of 1 minute angle. On the chart this is the letter o. A break across the top subtending a 1 minute angle makes the letter u; a similar break on the right side makes the letter c.

The addition of a 1 minute stem below and to the right of the o forms the letter q. A 1 minute stem below and to the left makes the letter p; above and to the right, the letter d, and above and to the left, the letter b.

The addition of a 1 minute stem below and to the right of the u forms a y.

A stem below and to the left of the c results in an f.

The combination of a break below with a stem above and to the left completes the letter h.

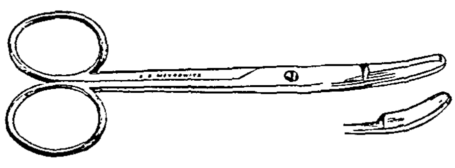
Several other letters might be used, but they would probably be too confusing. The letters are the result of one addition, one subtraction or a combination of one addition and one subtraction of 1 minute breaks and stems on a 3 minute base in the form of the letter o. This would appear to have accomplished the same two point separability as Landolt's broken circle and at the same time to have retained the practicability of the Snellen figures.

No claim is made that this is the perfect test type. I realize that some patients may have difficulty in interpreting the letters, though clearly seen. The dimensions of the component parts have not all been held to 1 minute. Some are 2 minutes, but none is higher. However, at the time this is the only combination of Landolt's and of Snellen's types that appears practicable, and it is submitted as such.

NEW SCISSORS FOR ENUCLEATION

Henry G. Wincor, M.D., New York

In operating for enucleation one is often disappointed at the small section of optic nerve obtained attached to the globe. Occasionally the nerve is cut at its insertion into the globe, so that no portion of the nerve is obtained for laboratory examination.



New scissors for enucleation.

The instrument pictured here was devised to enable one to obtain a good section of optic nerve. The elevation on the anterior blade is approximately 6 mm. When the scissors are inserted inside the capsule funnel and straddle the optic nerve, the act of closing the blades automatically raises the globe and puts the optic nerve on the stretch. Incidentally, the globe is protected from being cut or injured. The nerve is then cut by the lower blade.

The amount of nerve obtained is always equal to the height of the elevation on the upper blade and sometimes more. There should be no difficulty in inserting the scissors behind the globe. Grasping the globe with a forceps and rocking it sideways while inserting the blade will facilitate the introduction of the scissors.

These scissors have been tried out at various times on large eyes, on small atrophic globes, on phthisic globes and on children's eyes. In each instance a large section of nerve was obtained.

Ophthalmologic Reviews

EDITED BY DR. FRANCIS HEED ADLER

OCCUPATIONAL KERATITIDES AND CORNEAL DYSTROPHIES

M. DAVIDSON, M.D.
NEW YORK

The definition of a subacute or chronic occupational disease as distinguished from an acute industrial injury due to a single accident has been considerably broadened by legislatures. One therefore has to consider other occupational diseases of the eyes in addition to those previously recognized, namely, miners' nystagmus, glassblowers' cataract and other forms of radiation cataract and cataracts due to systemic poisons. The coverage of occupational diseases is extended not only in relation to the systemic poisons which may indirectly affect vision but in relation to certain substances which may affect the eyes directly, namely, dusts, fumes and oily particles. Those agents affecting the eyes directly may be described as giving rise to occupational keratitides and dystrophies. A clinical and medicolegal review of the subject is therefore presented.

A satisfactory etiologic classification of corneal diseases free from conflicting pathologic and anatomic-topographic criteria and morphologic terms, is as yet not available to the student. Duke-Elder's "Textbook of Ophthalmology"[1] still follows Fuchs'[2] clinical classification of corneal diseases into superficial and deep keratitides and dystrophies. Its artificiality is obvious from the facts that superficial punctate keratitis may be observed secondary to iridocyclitis and a deep involvement in the form of a disciform keratitis may be the end result of superficial herpes corneae. Both authors, moreover, deal with some keratitides under conjunctival diseases because of anatomic and practical considerations. Otchapovsky,[3] in a recent attempt at a classification of the keratitides, introduced a group of true keratoconjunctivitides, characterized by involvement of the limbic barrier, and divided them into nonulcerative chronic forms, with infiltration and vascularization, such

1. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2.

2. Fuchs, E.: Text-Book of Ophthalmology, ed. 7, Philadelphia, J. B. Lippincott Company, 1923.

3. Otchapovsky, S. V.: De la physiologie pathologique de la cornea, in Kewchariantz, A. G.: Sbornik Dolganova, Leningrad, Académie militaire de médicine RKKA de S. M. Kirov, 1936, p. 341.

as phlyctenulosis, trachoma and vernal catarrh, and ulcerative acute forms, without vascularization or necrosis but with erosions and related to dermatoses, such as herpes, eczema and rosacea keratoconjunctivitides. He therefore questioned the propriety of calling these conditions conjunctivitides. To classify corneal diseases into ectogenous and endogenous forms and those secondary to other diseases of the eye, as suggested by Fuchs and followed by Duke-Elder, is a mere approach to an etiologic classification, but it can hardly be considered as such. The slit lamp has made the classic clinical classification less and less tenable, since it enables one to make a more accurate localization of the depth of lesions and more accurate tracings of the path they take in their evolution.

A virus causation is being increasingly designated as the cause of a large group of corneal diseases. Grüter [4] ascribed a herpetic nature to dendritic, vesicular, bullous, disciform, superficial punctate, alphabet, reticular and interstitial keratitides and to relapsing erosions. Vazquez-Barriere,[5] on the basis of present knowledge, listed the following corneal diseases in the virus group: herpes corneae, herpes zoster ophthalmicus, superficial punctate keratitis, disciform keratitis of Fuchs, the metaherpetic keratitis of Vogt, keratitis nummularis, relapsing erosions and certain forms of deep keratitis. He found them characterized by vesiculation, ulceration, infiltration, lack of anchorage of epithelium to Bowman's membrane, rapid changes in structure, recurrence and hypesthesia. To these one may now add trachoma and some cases of endophthalmitis, according to Friedenwald and McKee.[6] Vitamin starvation is assigned an etiologic role in some keratitides, and allergies and endocrinopathies are increasingly being recognized as factors in others.

Dystrophies are somewhat better classified as senile, hereditary, secondary and occupational. Owing to the use of the slit lamp, occupational keratitides and dystrophies have been more and more frequently reported of late, in lieu of the diagnosis of conjunctivitis due to occupations.

The occupational keratitides as well as several other keratitides are treated by Fuchs [1] under diseases of the conjunctiva, and occupational conjunctivitis is defined as "an acute conjunctivitis found in certain industries where irritants—acrid vapors, liquids, or dustlike particles—get into the eyes either by accident or as part of the day's work." Under conditions allied to corneal dystrophies are further described changes

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4. Grüter, W.: Beiträge zum mikroskopischen Bild des Hornhautherpes, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **50**:223, 1934.

5. Vazquez-Barriere, A.: El virus herpetico en patologia ocular, Oftalmología **2**:16, 1936.

6. Friedenwald, J. S., and McKee, C. M.: Agent of Endophthalmitis, Am. J. Ophth. **21**:723 (July) 1938.

produced in the deeper layers of the cornea by the continuous action of various substances, such as lime, lead, silver, nitronaphthalin and iron (siderosis). Occupational keratitides are considered by Duke-Elder both under the conjunctivitides and under the keratitides. Under conjunctival diseases and allergies an extensive list of injurious substances is given, and a complete bibliography brings the subject up to date. The subject is further dealt with in Duke-Elder's text under superficial punctate keratitis, which is defined not as a disease entity but as a name embracing a large number of diseases. Under dystrophies further reference is made to electric ophthalmia and to traumatic band-shaped keratitis from mercury fumes and among hatters. There is a strikingly frequent reference to occupational injury or disease as an etiologic factor in the literature dealing with band-shaped keratitis.

The term superficial punctate keratitis, which Fuchs applied to a condition affecting the subepithelial layers only, is today applied with less rigor. Since the introduction of the slit lamp, it is being applied to lesions affecting the epithelial layer as well. Vogt [7] expressed the belief that the polymorphism of the epithelial keratitides is too great to permit a systematic classification and described a "keratitis epithelialis vesiculosa disseminata," a "keratitis epithelialis diffusa" and a "keratitis epithelialis marmorata." Duke-Elder [1] included some of the herpetic keratitides under superficial punctate keratitides. Thiel [8] described superficial punctate keratitis as a condition encountered in acute conjunctivitis, herpes and electric ophthalmia. While the lesions are not often mixed epithelial and subepithelial lesions, superficial punctate keratitis is a preferable term for the occupational keratitides, which embrace epithelial as well as subepithelial varieties.

OCCUPATIONAL SUPERFICIAL PUNCTATE KERATITIS

Occupational superficial punctate keratitis is not a serious condition per se. Except for the acute exacerbations and intermittent blurring of vision, visual acuity is rarely perceptibly affected. It, therefore, does not present a problem in workmen's compensation and is primarily of concern to the industrial hygienist and physician, because it presents a locus minoris resistentiae, influencing the effects of foreign bodies and erosions, their treatment and infections. The diagnosis of subacute or chronic conjunctivitis, a necessarily concomitant condition of superficial punctate keratitis and its exacerbations, should therefore be made by the plant physician with due consideration of a possible occupational factor. A keratitis is obviously a more serious condition for a worker to carry than a subacute or chronic conjunctivitis.

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7. Vogt, A.: Atlas der Spaltlampenmikroscopie, ed. 2, Berlin, Julius Springer, 1931, vol. 1.

8. Thiel, R.: Atlas der Augenkrankheiten, Leipzig, Georg Thieme, 1937.

The symptoms of superficial punctate keratitis, without the use of the slit lamp, may give rise to the diagnosis of acute, subacute or chronic conjunctivitis. Vogt [7] described the symptoms found in the epithelial keratitides in general as photophobia, blurring of vision, particularly after the eyes have been kept open for long periods and have become dry, and sometimes pain. Pick,[9] who dealt with an epidemic involving almost 50 per cent of the workers in a mushroom canning factory, listed the symptoms as photophobia, stabbing pain and blurring of vision, especially in the evening. Klein [10] reported severe blepharospasm and lacrimation among workers in the rayon industry. Rankine,[11] who reported 1,598 cases of superficial punctate keratitis observed in two years among workers in the acid house in the rayon industry, listed the symptoms as blurred vision and halos, grittiness, photophobia, lacrimation, blepharospasm, aching back of the eyes and headaches. Bücklers,[12] who reported a series of cases of superficial punctate keratitis in workers with lacquers in the furniture and metal industries, referred to similar observations among workers in the straw hat industry. He described the symptoms as lacrimation, grittiness and burning and stabbing pains, with an increase toward the end of the day and working week and most marked symptoms during the winter months when the ventilation of workrooms is poorest. According to McDonald,[13] "the workers in the spinning room complain frequently of misty vision, halos about lights, pain in the eyeball and tearing. . . . The symptoms are much more acute, but fleeting (as compared with CS_2 [carbon disulfide]) and are usually confined to those working in the spinning, washing and reeling rooms, where some 20 per cent of the workers claimed losing some time from work because of 'sore eyes.'" Powell [14] described a uniocular conjunctivitis from peat dust, observed in 105 agricultural workers on reclaimed marsh lands in California, the symptoms consisting of profuse lacrimation, intense pain and photophobia. Anneberg [15] reported a keratitis from exposure to pigweed and redroot and

9. Pick, L.: Augen- und Schleimhauterkrankungen durch Morchelausdünstungen, Ztschr. f. Augenh. **61**:325 (April) 1927.

10. Klein, E.: Les lésions oculaires dans les fabriques de soie artificielle, Arch. d'opht. **45**:686 (Nov.) 1928.

11. Rankine, D.: Artificial Silk Keratitis, Brit. M. J. **2**:6 (July 4) 1936.

12. Bücklers, M.: Tröpfchenförmige Niederschläge auf der Hornhautoberfläche bei Möbelarbeitern, Klin. Monatsbl. f. Augenh. **99**:676 (Nov.) 1937.

13. McDonald, R.: Ophthalmological Aspects, in Survey of Carbon Disulphide and Hydrogen Sulphide Hazards in the Viscose Rayon Industry, Bulletin 46, Occupational Disease Prevention Division, Commonwealth of Pennsylvania, Department of Labor and Industry, 1938, p. 38.

14. Powell, B. J., Jr.: Uniocular Conjunctivitis from Peat Dust, Am. J. Ophth. **17**:206 (March) 1934.

15. Anneberg, A. R.: Corneal Reaction to Weed Pollen, Am. J. Ophth. **21**:1265 (Nov.) 1938.

to other weeds among agricultural workers, with symptoms of itching, pain and foreign body sensation. All writers stress evidence of individual predisposition in the form of frequent recurrences in some persons and immunity of other neighboring workers. Thiel [16] also reported having frequently found keratitis punctata superficialis a problem in the sugar and rayon industries. The symptomatology is, therefore, rather uniform.

There is equal unanimity as to the biomicroscopic findings. Vogt [7] described the lesions as closely packed grayish points, commas or lines, from 0.1 to 0.02 mm. in size and even smaller, and seen only after staining with fluorescein; he also stated that they are sometimes seen as confluent lesions in the form of drops without infiltrates or vascularization. According to Pick,[9] the lesions do not stain during the acute stage of onset but later are demonstrable by staining as irregular points, lines and confluent lesions. Klein's [10] description of the biomicroscopic picture included chemosis, conjunctival hemorrhages, corneal edema, infiltrates and later erosions and sometimes confluent lesions. Rankine [11] found edema or irregular swelling of the corneal epithelium and desquamation of the epithelium, with a staining reaction to fluorescein. Bücklers [12] described the lesions as grayish transparent drops, about 0.04 mm. in diameter, staining only occasionally and visible only on transillumination but not directly with the slit lamp. Thiel [8] described the lesions of superficial punctate keratitis as minute gray, discrete and confluent lesions within and beneath the epithelium. Powell's [14] article does not include a description of the biomicroscopic corneal findings, and corneal lesions are not established but are not excluded. Gutmann [17] reported stippling of the cornea and infiltrates among workers in the rayon industry. Rochat [18] reported erosions which stained with fluorescein among workers in the sugar industry. Thiel [8] reported erosions and vesicles also among workers in the sugar industry. McDonald [13] reported fine blisters in the epithelium in the acute stage. The slit lamp picture is, therefore, also rather uniform and is illustrated by drawings in many of the papers cited; it also agrees with that observed by me. Anneberg [15] found conjunctival hyperemia, miosis and large corneal subepithelial infiltrates with no staining reaction to fluorescein. Among other findings, Rankine mentioned a garlic breath, and McDonald, loss of the "corneal" reflex in 50 per cent of his cases among workers in the rayon industry. However, he stated that he was not sure whether to attribute the symptom to exposure to hydrogen

16. Thiel, R.: Keratitis traumatica als Betriebsunfall in Zuckerfabriken, Klin. Monatsbl. f. Augenh. **81**:835 (Dec.) 1928.

17. Gutmann, A.: Augenentzündungen bei Kunstseidefabrikarbeitern, abstracted, Klin. Monatsbl. f. Augenh. **81**:727 (Nov.) 1928.

18. Rochat, G. F.: Schädigung der Hornhaut durch Schwefelwasserstoff, Klin. Monatsbl. f. Augenh. **70**:152, 1923.

sulfide or to intoxication from carbon disulfide, to which the workers are also exposed. It is interesting that Gundersen [19] reported not having observed any vesicles in the herpetic corneal diseases he studied.

For the demonstration of lesions of superficial punctate keratitis, I prefer a 2 per cent solution of mercurochrome, which is less diffusible than fluorescein and provides a better contrast between the lesion and the surrounding area, and slit lamp examination by transillumination from the iris with the microscope focused on the cornea. While the normal cornea shows no epithelial desquamation, the normal conjunctiva appears to be continuously desquamating when stained with mercurochrome, particularly the exposed and lower bulbar conjunctiva. Because of its rapidity, this method should be superior to either corneal esthesiometry or "corneal" reflex testing for mass examination of workers in a plant.

The outstanding offending agents appear to be hydrogen sulfide, principally in the rayon and sugar industries, and benzene and similar solvents and diluents of varnishes and shellacs and lacquers. Likhtner [20] reported on the superficial punctate keratitis observed in persons employed in the cracking process of coal rich in sulfur; he considered a concentration of 1:1,000,000 of hydrogen sulfide as the limit of permissibility. Pick [9] mentioned drops of helvellic acid as an offending agent. Both Klein [10] and Bücklers [12] referred to drops of chemicals on the cornea, and the drops scattered by spinning and spraying rather than the gases involved in the industrial processes are stressed as the factor. According to McDonald, hydrogen sulfide droplets of the acid bath are responsible for keratitis among workers in the rayon industry; he found the condition more marked in damp weather and stated that a concentration of 10:1,000,000 is permissible. Thiel [16] also arrived at the conclusion that hydrogen sulfide is responsible for the keratitis, but he considered the possibility of its conversion into an organic sulfur compound before becoming toxic or of the keratitis being secondary to a dystrophic corneal process caused by the exposure. In Powell's report,[14] however, silica spicules and acids salts are mentioned. Other of the more important agents considered in textbooks and in the literature are hydrogen fluoride, tar pitch, mercury and silver sublimate, certain kinds of woods and lead nitrate. Duke-Elder's [1] list includes many chemicals and animal and vegetable dusts and allergens, such as atropine and pontocaine. This variety of occupational superficial punctate keratitis may be designated as the subacute variety, although some writers consider it acute because of exacerbations. The patients all get

19. Gundersen, T.: Herpes Corneae, with Special Reference to Its Treatment with a Strong Solution of Iodine, Arch. Ophth. **15**:225 (Feb.) 1936.

20. Likhtner, V. A.: Eye Injuries Due to Hydrogen Sulfide in a Petroleum Refining Plant, Vestnik oftal. **11**:256, 1937.

well with change of occupation, and there is restitutio ad integrum in from seven to ten days.

At the New York State Bureau of Workmen's Compensation I have observed only a few cases of the acute and subacute variety, caused by liquid drops in the air clinging to the cornea; the scarcity of such cases is probably due to the vigorously enforced safety measures and the transient nature of the condition as well as to the tendency on the part of the worker to invoke a single accident rather than an occupational disease, owing to the past limitations of the definition of occupational disease. The cases (except 1 in 1937) observed in the last six months occurred in the following persons: a shellac bleacher, a mixture of chlorine gas and sodium hydroxide being the contributing factor; a chemist, fumes of allyl alcohol and formic acid being the agent; a nickel polisher; a sugar crusher, raw sugar dust being the contributing factor; a leather worker, formaldehyde from leather shavings being the exciting agent, with a possibility of trachoma complicating the picture; and a brass filer with a pseudomembranous conjunctivitis and subconjunctival foreign bodies. The last-mentioned patient is still being studied.

SYMPTOMLESS OCCUPATIONAL SUPERFICIAL PUNCTATE KERATITIS

A symptomless form of superficial punctate keratitis which verges on a dystrophy was first reported by Samoilov [21] among lathe workers. That the superficial punctate keratitides of the herpetic group are followed by hypesthesia and even by hypotension is, of course, familiar. Gifford [22] reported on a mild form of epithelial dystrophy of the cornea, thought to be mainly senile, with hypesthesia. In his study of lathe workers, who presented an incidence of corneal scarring of 20 per cent, Samoilov reported a definite corneal hypesthesia amounting to 92 per cent, established by esthesiometric examination with von Frey hairs. Intravital staining with methylene blue showed not only epithelial erosions but disappearance of the normal end organs of the corneal nerve, which Samoilov stated can be abundantly seen in normal eyes by this method. The destruction of the end organs of the corneal nerve he regarded as evidence of a process reaching the subepithelial region. The corneas also showed a greater rate of desiccation than normal eyes. The condition was symptomless, except for pericorneal injection on manipulation of the lids over the eyes. Samoilov attributed the condition to constant bombardment of the corneas by sharp spicules of metal.

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21. Samoilov, A.: Professionalnaia patologia glaza, Obuch Institute for the Study of Occupational Diseases, Moscow, Izdatelstvo Mozdravotdiela, 1929.

22. Gifford, S. R.: The Mild Form of Epithelial Dystrophy of the Cornea, Arch. Ophth. 7:18 (Jan.) 1932.

Almost every stonecutter, grinder or sandblaster with an accidental injury appearing for ocular examination at the New York State Bureau of Workmen's Compensation has shown numerous bilateral, minute, irregularly shaped and sharp-cornered scars, diffusely scattered throughout the cornea, and often still more minute glistening foreign bodies embedded in the scars. There are no complaints by the claimants other than those referable to the accident and the eye involved. Many of them also exhibit a generally bilateral but sometimes unilateral corneal hypesthesia and minute epithelial erosions; neither type, however, has had any definite relation to the number of scars. Since this condition was not legally an occupational disease and was moreover only rarely accompanied by any appreciable loss of visual acuity and was not compensable until recently, no particular record was kept of the cases except to call attention to the existence of the condition and the possible parallelism with pulmonary silicosis.[23] There are, moreover, few stonecutters in the city of New York, and only about a dozen may be seen at the bureau in the course of a year. The three stonecutters and one grinder encountered in the last six months all had vision of 20/20 and minor ocular injuries, with no complaint except that referable to the eye involved. The stonecutters had worked thirty-four, forty and forty-five years, respectively, at their trade and the grinder, fourteen years; it is probable that younger workers are not affected. All showed scars, hypesthesia and erosions except one, who had scars only.

The influence of an occupational corneal dystrophy on the evolution and development of disciform keratitis, generally characterized by vascularization from the intrascleral plexus by a single vessel, is illustrated by the case of a stonecutter observed at the bureau. This claimant has been a stonecutter for ten years, then a policeman for twenty-five years, and then a stonecutter again for four years. An apparently insignificant foreign body for which he did not seek attention for four days gave rise to a deep ulcer by that time, followed by hypertension, which necessitated paracentesis, and a disciform keratitis as a sequel. The presence of "stonecutters' cornea" in the fellow eye and complete anesthesia make for the probability that the bilateral corneal dystrophy was the result of the original ten years' exposure rather than the exposure of the last four years under modern safety conditions, thus justifying the application of the term dystrophy to the condition.

OCCUPATIONAL DYSTROPHIES

True occupational dystrophies, such as are reported in textbooks and in the literature, have not been observed. In cases of argyrosis

23. Davidson, M.: Silicosis Corneae, Am. J. Ophth. **19**:896 (Oct.) 1936.

Stein,[24] Subal[25] and Blind[26] reported grayish deep opacities and a network of grayish yellow lines in Descemet's membrane. Harrison[27] described a primary zonular opacity of the cornea in a hatter due to mercury fumes.

Zonular, or ribbon-shaped or band-shaped, opacity or calcareous band of cornea was described by Fuchs[2] as the most frequent of the pathologic type of dystrophies, developing because of intraocular disease, in senile persons or because of external injurious influences to which corneas have been subjected in the region of the palpebral fissures.

Michail[28] expressed the belief that an angioneuritis, a sympatheticotonia of the surface vessels or a dilatation of subepithelial vessels of conjunctiva is the cause of calcium deposition and of band-shaped keratitis. Comberg[29] explained the site and development of band-shaped keratitis and relapsing erosions by a tendency of epithelial regeneration to start simultaneously from above and below and to result in an overgrowth at or near the meeting point with the horizontal meridian.

Cases of dystrophies sequels of accidents are, however, encountered. Three patients with lime burns of long standing were seen during the past year. Examination proved that a degenerative process goes on in the cornea with lime incrustation in the course of years. One patient was examined at the New York State Bureau of Workmen's Compensation in connection with a lime burn of the left eye. There was a history of a lime burn in the right eye some twenty years before; the eye showed, aside from a lime incrustation in the upper corneal region, a band-shaped lime incrustation in the lower corneal region with typical etched subepithelial lines and numerous chalky white calculi lying at different levels in the corneal stroma as far back as Descemet's membrane, where they were star shaped and lying in round clearings in a homogeneously somewhat opaque membrane. In another patient the same transport of lime had progressed to deposit calculi to about the middle of the cornea in the course of two years. In the third this was observed beginning within six months after a lime burn. In addition, superficial punctate keratitis has been observed by staining and by bio-

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24. Stein, R.: Berufsargyrosis der Hornhaut, Klin. Monatsbl. f. Augenh. **81:** 731 (Nov.) 1928.

25. Subal: Berufschädigungen der Bindehaut und Hornhaut durch Silber, Klin. Monatsbl. f. Augenh. **68:**647 (April-May) 1922.

26. Blind: Ein Fall von Gewerbeargyrosis des Auges, Klin. Monatsbl. f. Augenh. **76:**884 (June) 1925.

27. Harrison, W. J.: Primary Zonular Opacity of the Cornea, Arch. Ophth. **16:**469 (Sept.) 1936.

28. Michail, D.: Dégénérescence calcaire primitive de la cornée, Arch. d'opht. **52:**247 (April) 1935.

29. Comberg, W.: Experimentelles und Klinisches über das Hornhautepithel, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **50:**209, 1934.

microscopic examination in persons with an apparently long cured lime burn who complain of recurring redness of the eye.

Wagenmann [30] credited De Gauvea (1869) and Andreae (1898) with observations of the slow transport of lime as far as Descemet's membrane in cases of lime burns; he also quoted Gühman to the effect that there results in the presence of lime incrustation a double fluid current from the lime deposit and the aqueous, with precipitation of lime salts. Thies [31] called attention to the diffusion of both albumin-dissolving alkalis and albumin-precipitating acids into the aqueous in cases of lime burns, with resulting changes in the iris, lens, ciliary body and vitreous and hypertension. These manifestations may be delayed and present problems in the differential diagnosis between injury and disease.

TRAUMATIC FACTOR IN THE HERPETIC GROUP OF CORNEAL DISEASES

The medicolegal problems involved in the occupational keratitides and dystrophies center around the problem of the traumatic factor in the etiology of the herpetic group of corneal diseases.

While herpes corneae and herpes zoster ophthalmicus are almost always unilateral conditions with rapid morphologic changes, bilaterality, steadiness of the morphologic picture and particularly absence of dendritic transformations characterize the occupational diseases. Powell's group of cases of unilateral involvement is to be explained by the possibility of unilateral voluntary closure of the eye permitted by the situation.

There is, however, a traumatic factor in herpetic disease to be considered per se. Schmidt [32] quoted Favery to the effect that trauma is a factor in the origin of herpes corneae in only 1 per cent of cases. In a study of a large group of cases of corneal disease of a herpetic nature Schmidt himself did not find trauma a factor in any of the cases of dendritic keratitis; a traumatic origin was discovered in only 1.8 per cent of the cases of superficial punctate keratitis but in 17.4 per cent of the cases of vesicular keratitis and in 20 per cent of the cases of relapsing erosions. He suggested that an interval of from two to five days should elapse between trauma and the onset of disease before a causal relationship is considered. Schieck [33] pointed out that since Busacca has demonstrated the herpes virus in unaffected conjunctivas and since the transmission of herpes to the rabbit's cornea is done by means of direct inoculation into

30. Wagenmann, A.: Die Verletzungen des Auges, in Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 3, Leipzig, Wilhelm Engelmann, 1915-1925.

31. Thies, O.: Die Verlätzungen des Auges, Stuttgart, Ferdinand Enke, 1938.

32. Schmidt, K.: Herpes corneae und Trauma, Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **50**:217, 1934.

33. Schieck, in discussion on Schmidt,[32] p. 231.

the cornea, one should feel freer in accepting a traumatic factor and that one day is to be considered as the period of incubation. Gundersen,[19] in his study of 250 cases of herpes corneae, reported that the origin was probably traumatic in 5.2 per cent; he stated that the "most that can be said is that when the disease closely follows a definite trauma, it is probable that the trauma is the exciting cause." Ammann [34] reported cases of traumatic herpes corneae and 6 cases of keratitis superficialis interstitialis traumatica, the description of which corresponds to what one would call disciform keratitis, following apparently trivial corneal injuries. The same has also been seen by me. Ammann [35] also reported a group of 10 cases of herpes zoster ophthalmicus traumaticus from the literature. At the Bureau of Workmen's Compensation the percentage of cases of traumatic herpes cornea would of course be much higher than the foregoing figures would indicate.

SUMMARY

Occupational superficial punctate keratitis and dystrophy are often diagnosed as subacute or chronic conjunctivitis and as band-shaped keratitis and the true nature of the conditions overlooked without the use of the slit lamp.

Three types of this form of keratitis may be recognized. The first is an acute or subacute occupational superficial punctate keratitis, apparently due to particulate matter in spraying processes which clings to the corneal epithelium rather than to gases. This type is not important and to judge from the small number of cases seen, is evidently successfully combated in the state of New York by the safety measures employed. The second type is a chronic occupational superficial punctate keratitis verging on a dystrophy and apparently due to hard and sharp spicules penetrating the cornea, such as silica or metals. This type is more important because of the hypesthesia, but is also not common and is seen only among the older workers, being principally due to exposures in the presafety era. The third type is an occupational band-shaped keratitis seen at the bureau in the form of a post-traumatic lime band. Medicolegally, an interval of from one to five days is to be considered a requisite for a causal relation between a corneal injury and a herpetic corneal disease.

The problem of prevention of occupational keratitides and dystrophies is essentially one of safety engineering.

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34. Ammann, E.: Herpes traumaticus und Art. 91 K. U. G., Schweiz. med. Wchnschr. **63**:809 (Aug. 19) 1933; Keratitis superficialis-interstitialis traumatica, Klin. Monatsbl. f. Augenh. **91**:253 (Aug.) 1933.

35. Ammann, E.: Herpes zoster ophthalmicus traumaticus, Klin. Monatsbl. f. Augenh. **95**:793 (Dec.) 1935.

News and Notes

SOCIETY NEWS

American Ophthalmological Society—The American Ophthalmological Society will celebrate the seventy-fifth anniversary of its founding at a meeting to be held at the Homestead, Hot Springs, Va., June 5 to 7. In memory of the distinguished ophthalmologists who were instrumental in organizing the first society of medical specialists in the United States, the program of the usual three day meeting has been augmented to include a banquet, at which papers bearing on the relation of this society to the development of ophthalmic science will be read. At the banquet the history of the society will be presented in a paper by Dr. Harry Friedenwald, of Baltimore, who has been a member since 1894. Mr. H. M. Traquair, of Edinburgh, Scotland, will be the invited guest. At the first meeting of the society, held on June 7, 1864, at the New York Eye Infirmary, Dr. Edward Delafield, of New York, presided. He was reelected to serve as president for four successive terms. A fitting tribute to the society's first president will be presented at the banquet by Dr. Bernard Samuels, who will read a paper on the life and influence of Dr. Delafield.

International Association for the Prevention of Blindness.—The International Association for the Prevention of Blindness, which maintains a secretariat in Paris, will hold its next annual meeting in London, England, April 19, 1939. The principal topic for discussion will be "The Application of the Credé Method for Prevention of Blindness in Various Countries." The United States will be represented by Dr. Conrad Berens, of New York, a member of the board of directors of the National Society for the Prevention of Blindness; Dr. Park Lewis, of Buffalo, vice president of both the International Association and the American National Society, and Dr. Harry S. Gradle, of Chicago, vice president of the Illinois Society for the Prevention of Blindness.

North of England Ophthalmological Society.—The North of England Ophthalmological Society will sponsor an ophthalmologic tour, visiting New York, Philadelphia and Washington. The group is scheduled to reach New York on Sunday, April 30, remaining there until Friday, May 5. The visiting ophthalmologists will spend Saturday and Sunday, May 6 and 7, in Philadelphia and May 8 and 9 in Washington and will leave New York on Wednesday, May 10. Optional extensions for sight-seeing are available. The tour will be conducted by Thomas Cook & Son, Ltd.

Puget Sound Academy of Ophthalmology and Otolaryngology.—Dr. J. Edward Clark, Seattle, Wash., was recently elected president of the Puget Sound Academy of Ophthalmology and Otolaryngology; Dr. W. A. Cameron, Tacoma, Wash., was elected vice president and Dr. Purman Dorman, Seattle, secretary-treasurer.

German Ophthalmological Society.—The next meeting of the German Ophthalmological Society will be held in Heidelberg on Sept. 4-6, 1939. One principal topic will be considered, and there will be many papers on individual subjects. The Graefe medal will be awarded to a representative of the family of the late Jules Gonin.

GENERAL NEWS

Sight-Saving Classes.—The National Society for the Prevention of Blindness has announced that it is cooperating with the following colleges and universities in offering at their 1939 summer sessions courses for the preparation of teachers and supervisors of sight-saving classes:

Western Reserve University, Cleveland, June 19 to July 28. Director of the course: Miss Olive S. Peck, supervisor of Braille and sight-saving classes, Board of Education, Cleveland.

State Teachers College, Buffalo, June 26 to August 4 (dates tentative). Director of the course: Miss Agnes Reuter, Department of Special Education, Buffalo Public Schools.

State Teachers College, Milwaukee, June 26 to August 4. Director of the course: Miss Marguerite L. Kastrup, supervisor of Braille and sight-saving classes for Northern Ohio, Cleveland.

University of California, Los Angeles, June 26 to August 4. Director of the course: Miss Frances Blend, principal of sight-saving classes, Los Angeles City Schools.

Wayne University, Detroit, June 26 to August 4 (elementary and advanced courses). Director of the elementary course: Mrs. Gladys Dunlop Matlock, Detroit. Director of the advanced course: Mrs Winifred Hathaway, associate director, National Society for the Prevention of Blindness, New York.

Details regarding the courses may be obtained from the university or college or from the director in charge of the course.

Ophthalmologica, International Journal of Ophthalmology.—*Ophthalmologica* is the continuation of the *Zeitschrift für Augenheilkunde*. It now appears as an international journal of ophthalmology. Prof. A. Brückner, of Basel, Switzerland, and Prof. H. Weve, of Utrecht, Netherlands, are the new editors. There is an imposing list of co-editors who represent practically all countries in the world. The journal will continue to be published monthly, by S. Karger, in Basel; six numbers will make a volume, the price being 30 Swiss francs ($6).

The original articles are to appear in any one of three languages, German, English or French, with summaries in all three languages. A report on ophthalmologic literature, society reports, a chapter on diagnosis and therapy and book reviews complete the number.

PERSONAL

Dr. Phillips Thygeson has been appointed executive officer of the department of ophthalmology in Columbia University's College of Physicians and Surgeons. Dr. John H. Dunnington has been made clinical director. Both Dr. Thygeson and Dr. Dunnington were promoted to full professorships.

Obituaries

RICHARD GREEFF, M.D.
1862-1938

Richard Greeff, the son of a professor of zoology in the University of Bonn, received an early introduction to the natural sciences. He obtained his ophthalmologic training from Schweigger and was appointed professor of ophthalmology in the University of Berlin. He was entrusted with the formation of the ophthalmic department at the Charité Hospital in Berlin in 1897, which he conducted until the time of retirement on account of age limit in 1927. During these years the department grew steadily, and Greeff established his reputation as teacher, clinician and scientist. His course on the pathology of the eye was popular and attracted many young ophthalmologists from all parts of the world. His histologic investigations began with the structure of the retina, in which he employed the methods of Ramón y Cajal. He was one of the first ophthalmologists to write a textbook on the pathology of the eye. His clinical investigations were concentrated on trachoma, and he was one of the discoverers of the inclusion body.

Of marked artistic leaning, he repeatedly visited the art treasures of Italy and Spain. There he discovered ophthalmologic points of interest in the works of art, such as Rembrandt's portrayal of Tobias' blindness and recovery, which he described in an interesting monograph. Greeff made an important collection of eye glasses and spectacles, to which were added ophthalmoscopes, surgical instruments and memorabilia pertaining to von Graefe. These are all on exhibition in the Kaiserin-Friedrich Museum in Berlin.

ARNOLD KNAPP.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

TOPOGRAPHY OF THE VENAE VORTICOSAE. G. BASILE, Ann. di ottal. e clin. ocul. 66: 100 (Feb.) 1938.

Because of the necessity for locating the venae vorticosae during operations for retinal detachment, the author studied their location in 39 eyes of cadavers. The eyes were injected to normal tension with a fine needle, and measurements were made of three factors relating to each vein: (1) the distance from the limbus to the center of the insertion of the superior and inferior rectus muscles, (2) the distance from the limbus to the scleral outlet of each vein and (3) the distance between the outlet of each vein and the center of the insertion of the superior rectus muscle for the two upper veins and of the inferior rectus muscle for the two lower veins. A triangle was formed, therefore, uniting the center of the cornea with the venous outlet and the center of the nearest insertion of the rectus muscle. The angle of the two lines with the corneal vertex was calculated for each vein, and graphs were made showing the relative frequency of the various angles at which the veins were found.

The distance of the superior temporal vein from the limbus varied from 16 to 19.8 mm.; that of the superior nasal vein, from 16 to 20.5 mm.; that of the inferior temporal vein, from 15.5 to 19.5 mm., and that of the inferior nasal vein, from 15 to 19 mm. The veins emerged most frequently along a meridian nearer to the vertical than to the horizontal meridian, the angle with the vertical meridian being most frequently between 25 and 30 degrees.

S. R. GIFFORD.

Bacteriology and Serology

ALLERGIC OCULAR REACTIONS. V. SPADAVECCHIA, Ann. di ottal. e clin. ocul. 66: 241 (April) 1938.

The phenomenon of Auer was produced in rabbits by sensitizing them to horse serum and after a suitable period injecting a large dose of the serum (10 cc.) intraperitoneally, followed by the instillation of an irritating chemical in the eye. When 2 drops of chloroform was employed, the initial period of hyperemia and edema due to the chemical was followed after several days by severe iritis, which persisted till the eighth day. This reaction was absent in the 2 control animals, 1 of which was sensitized but was not given a toxic dose of serum, while the other received the toxic dose without previous sensitization. The iritis which occurred in the sensitized animals which were given a second injection of serum must be considered an allergic phenomenon. The irritating chemical, by producing hyperemia and increasing capillary permeability, produced fixation of the heterologous protein in the ocular

tissues, resulting in an intense local reaction. Later instillations of chloroform in sensitized rabbits produced much slighter reactions. When the chloroform was diluted with two parts of liquid petrolatum, the difference between the control and the sensitized animals was more clearcut, the control showing a slight and brief reaction, while the sensitized animal showed a dense area of central corneal infiltrations with subsequent vascularization, accompanied by iritis, which persisted for a month.

When other irritants, such as histamine and uric acid, were employed, inflammation of the conjunctiva and in some cases of the iris resulted in the sensitized animals. The instillation of horse serum in sensitized animals produced only a slight and brief reaction, while its subcutaneous or retrobulbar injection produced marked swellings, which persisted for a number of days (phenomenon of Arthus). In all the experiments the most severe reactions were produced in animals which were sensitized and after an interval given a large dose of serum. Less intense reactions were seen in animals which were sensitized but were not given a toxic dose of serum at the time the chemical was applied.

S. R. Gifford.

Biochemistry

Occurrence of Acetylcholine Esterase in the Aqueous Humor and Vitreous Body. Börje Uvnäs and Herbert Wolff, Acta ophth. **16**: 157, 1938.

The authors, using the eyes of cattle and of horses, studied the hydrolytic action of aqueous and vitreous on acetylcholine. The method adopted involves colorimetry and is a modification of the technic for the estimation of acetylcholine esterase in blood serum. The article is summarized as follows:

Practically no acetylcholine esterase could normally be detected in the aqueous of bovine eyes. Only under certain conditions, viz., after postmortem changes, and more especially after a previous partial emptying of the anterior chamber, could enzyme activity be detected.

Considerable quantities of acetylcholine esterase were found in the vitreous of bovine eyes. The esterase activity of the vitreous corresponds approximately to one eighth of that found in blood from man and horse. In vitreous from the horse the activity of acetylcholine esterase was found to be much less.

The enzyme activity that appears under certain conditions in aqueous may conceivably be derived from inflowing or indrawn vitreous.

O. P. Perkins.

Conjunctiva

Treatment of Gonorrheal Conjunctivitis with Sulfanilamide. A. Magitot, A. Dubois-Poulsen and Y. Geffroy, Bull. Soc. d'opht. de Paris **50**: 82 (Feb.) 1938.

The literature and history of the drug are reviewed. Five cases are cited in some detail. These cases do not warrant precise conclusions but point the way to miraculous cures of this disease. The question of dosage is extremely important. The authors believe that the rule of

1 Gm. of the drug to every kilogram of body weight, as proposed by Schwentker and Clason, should be adopted. Cyanosis, dermatoses and anemia are the complications that should be contraindications for continued treatment.

L. L. MAYER.

CHEMOTHERAPY OF BLENNORRHAGIC CONJUNCTIVITIS WITH SULFANILAMIDE. R. PAGES and J. DUGUET, Bull. Soc. d'opht. de Paris **50:** 94 (Feb.) 1938.

Three cases of blennorrhagic conjunctivitis in which sulfanilamide was given are reported in detail. Outstanding benefits from this form of therapy are loss of photophobia, disappearance of pain, failure to secure positive cultures in short periods after treatment is begun and lessening of the purulent secretion. Local treatment should be continued in connection with the drug. The drug seems specific.

L. L. MAYER.

PARINAUD'S CONJUNCTIVITIS AND PAROTITIS (RELATION TO HEERFORDT'S SYNDROME). C. THOMAS, Bull. Soc. d'opht. de Paris **50:** 382 (June) 1938.

Since 1889, when Parinaud described "a conjunctival infection of animal origin," the etiology of this condition has been much discussed. Numerous organisms have been described as causing preauricular adenitis and conjunctivitis. A recent patient of Thomas was given a thorough bacteriologic examination by Professor de Lavergne, of Nancy, France. The lesion was a monopalpebral granuloma of the conjunctiva with preauricular adenopathy, which receded in a number of weeks of its own accord. All tests for organisms and general disease were negative. Biopsy of the glandular tissue and the granulomatous conjunctiva revealed changes similar to those reported by Besnier, Böeck and Schaumann. Thomas feels that the condition is perhaps a more localized phase of the uveoparotitis of Heerfordt. His reasons are as follows: 1. The conjunctival granulations are similar to those described by Toulant and Morard in Heerfordt's disease. 2. Tumefaction of the preauricular gland is similar. 3. The evolution and spontaneous healing are similar, except that uveoparotitis may last a little longer. 4. Histologically, a benign lymphogranuloma exists in each disease.

L. L. MAYER.

Congenital Anomalies

ANIRIDIA CONGENITA, IRIDEREMIA. E. M. NEHER, Am. J. Ophth. **21:** 293 (March) 1938.

After a discussion of aniridia and a presentation of genealogic studies, Neher gives the following summary:

"The part of the aniridic family tree upon which accurate data could be obtained has verified the Mendelian inheritance law; that is, one half of the offspring possess the dominant character—aniridia—while the

other half possess a normal iris. Furthermore, when the aniridic offspring are mated with normal individuals and have children, 50 per cent are aniridic and the remainder have a normal iris; but the normal offspring who marry normal individuals never have borne children with aniridia. The amount of iris present varies from complete aniridia to a small coloboma of the iris."

W. S. REESE.

Cornea and Sclera

SYNDROME OF BLUE SCLEROTICS (VAN DER HOEVE). J. S. CHARAMIS, Ann. d'ocul. **175**: 738 (Oct.) 1938.

The author states that for the first time in Greece it has been possible to study four generations in a family many members of which presented a van der Hoeve syndrome, either partial or complete.

This syndrome presents three classic symptoms: a blue sclera, multiple fractures and otosclerosis. In 1829 Lobstein first reported the constitutional predisposition of certain persons to fractures. Eddowes in 1901 reported that a blue sclera is often present in such persons. He also demonstrated the familial and hereditary character of the condition. In 1916 van der Hoeve and Klein added deafness as the third principal symptom. Practically all authors are agreed that the condition is a familial one and due to heredity.

It was seen in the family here studied that persons in perfect health had descendants who presented in part the syndrome in question. Certain families may present one of the three symptoms, such as deafness or a blue sclera or bone fragility, or two of these symptoms may be present. In the family here described the males seemed to be more afflicted than the females.

A description of the four generations is given in detail.

S. H. MCKEE.

Experimental Pathology

RETURN OF VISION AND OTHER OBSERVATIONS IN GRAFTED VERTEBRATE EYES. L. S. STONE, Am. J. Ophth. **21**: 1 (Jan.) 1938.

On the basis of this interesting study of grafted vertebrate eyes, Stone presents the following discussion and conclusions:

"It is clear that the functional eye of the urodele can be readily grafted, even several times, with complete recovery of all its functions so long as it is placed in its normal environment, the orbit. It is also obvious that the nature of the degenerative changes that take place in the grafted eye and the amount of regeneration required to bring about recovery depend upon the age of the host. The ability of the young eye to undergo transplantation with so little subsequent change in its structure, and the power exhibited by the adult eye to regenerate a new lens and retina after considerable destruction explain the high percentage of success in these experiments. The homoplastic transplantations seem to be about as successful as the autoplastic (reimplanted) graft. Even between closely allied species (Stone, '30) the

heteroplastic grafts are equally tolerated. Between the adults of more distantly related species (A. punctatum and T. viridescens) there appears to be incompatibility between host and graft tissues. This is not entirely mutual, however, because the Amblystoma eye upon Triturus can remain in good condition for several months. The reverse is not true.

"It remains to be seen whether or not there are any groups of vertebrates in which the eye can be so successfully grafted as in the amphibians. The works of Blatt ('24) and Ask and Andersson ('27) in grafted eyes, and the experiments of Matthews ('33) on cutting the optic nerve seem to give some evidence that the power of restitution in the operated eye in some of the fishes is not so great as in amphibians. The experimental work that we have begun in this class of vertebrates has also given us information that in Fundulus this is quite true. What differences there will be found in the various groups of fishes awaits further experimentation. There is no evidence existing at the present time.

"Among the other classes of vertebrates there is also no satisfactory information on this score. What little has been done tends to indicate negative results in the grafting of the eye in the higher forms, for in them regeneration of lost parts in the more specialized tissues seems to be rather poorly developed. Favorable findings in the grafted eye of the rat were published by Koppanyi ('23) but these results do not seem to have been corroborated. Our own unpublished experiments on more than 100 rats of various ages after birth indicate that anatomical healing in the grafted eye of the white rat is difficult to obtain. A reimplanted eye existing for about four months, one fourth of its original size, represented the best results in our series. The retina degenerated without the slightest evidence of recovery. However, the grafting of the eye of the mammal in general has not been investigated sufficiently to give an idea how much can be done. Therefore the results so far do not necessarily mean that better success is not possible in some other mammal.

"The size of the eye and the part it plays in delaying the return of circulation to distant parts of the bulb is no doubt one of the important features which extends the degenerative changes to the point where restitution becomes impossible. It is quite possible that in only those animals which exhibit exceptional powers of regeneration can we expect the eye to survive an operation such as grafting."

W. S. Reese.

General Diseases

Clinical and Pathologic Changes in Brucellosis. M. S. Nikolaeva, Vestnik oftal. **11:** 187, 1937.

A review of the historical, geographic and clinical data concerning ocular lesions in cases of brucellosis is given. The serologic and allergic tests are important in establishing a correct diagnosis. Two cases are reported by Nikolaeva. In 1 of these the patient, a man aged 29,

presented a deep keratitis and uveitis of both eyes with a subfebrile temperature. Wright's reaction of the blood was positive in dilutions of 1:50 and 1:200; Burnet's reaction was also positive, so that the diagnosis of a mild brucellosis was made. In the second case the patient, a man aged 60, presented a severe "typhoid-like" condition with a number of complications and a markedly positive Wright's reaction of the blood (dilution, 1:500). There were hemorrhages in the anterior chamber and in the vitreous of the right eye. In the left eye there was total ophthalmoplegia. Both eyes were removed at autopsy and examined. Six photomicrographs illustrate the pathologic changes in the eyes. Nikolaeva arrives at the following conclusions:

1. The ocular lesions in brucellosis vary greatly. There may be a superficial or a deep keratitis, iridocyclitis or inflammation of the optic nerve.

2. The process is localized more frequently in the anterior part of the uvea. The clinical picture is not typical; the diagnosis is therefore difficult and can be established more accurately by the means of serologic and allergic reactions.

3. The paralysis of the oculomotor nerve is associated with severe lesions in the central nervous system.

4. Ocular lesions are observed in "ambulatory" patients with mild brucellosis and in those with the severe "typhoid-like" type of brucellosis.

5. The pathologic changes of the eye in cases of brucellosis give a picture of diffuse, nonspecific, chronic inflammation with a tendency to hemorrhages and a mild degree of exudates without the formation of specific granulomas. (This is confirmed by experimental brucellosis.) A detachment of nonpigmented epithelium of the ciliary body was observed which resembled the process of sympathetic ophthalmia.

O. Sitchevska.

General Pathology

Senile Changes and Degenerations of the Human Eye. B. Rones, Am. J. Ophth. **21:** 239 (March) 1938.

This interesting article does not lend itself to abstracting. Rones discusses the senile changes in the different structures of the eye and their possible clinical applications. He gives the following summary:

"Analyzing the factors contributing to the diversity of senile changes in the ocular tissues leads to the conclusion that the vascular changes are of fundamental importance. Impaired nutrition resulting from this will explain the deposition of fat globules in the various structures. It is also well known that initial proliferative changes leading to subsequent degenerations are attributable to faulty circulation. The old adage that 'a man is as old as his arteries' can thus be also applicable to the changes that occur in the eye during advancing years." W. S. Reese.

Glaucoma

RECENT ADVANCES IN THE SURGERY OF CHRONIC GLAUCOMA. O. BARKAN, Am. J. Ophth. **20:** 1237 (Dec.) 1937.

Following a discussion relative to his recently presented operation, Barkan gives the following summary:

"1. The procedure of opening Schlemm's canal under direct vision involves a new principle in the surgery of glaucoma in that a hitherto hidden area of the inside of the eyeball (Schlemm's canal and the angle of the anterior chamber) is made visible during operation.

"2. The surgeon can see the area of blockage during the operation and can actually watch and guide his instrument in restoring the normal direction to the circulation of intraocular fluid.

"3. The operation is without danger when the proper technique is used and has proved successful when certain preoperative indications (biomicroscopic diagnosis) have been fulfilled.

"4. The results are predictable and appear to be permanent. However, a much longer period of observation will be necessary to give a definite answer to the question of permanency of results.

"5. The operation is especially indicated in chronic simple glaucoma (type 1 of the writer). It is equally successful in certain cases of secondary glaucoma and gives promise of being applicable also in certain other cases of primary glaucoma.

"6. The pathologic anatomic entity described by the writer as chronic glaucoma, type 1, appears to cover "chronic simple glaucoma," and "anterior glaucoma."

"7. Certain conclusions in regard to the etiology and symptomatology of chronic simple glaucoma may be fairly drawn.

"8. Biomicroscopy of the angle of the anterior chamber is a useful adjunct to diagnosis and often enables an earlier diagnosis of glaucoma than has hitherto been possible. It would seem to constitute a considerable advance in the management of glaucoma.

"9. The operation of opening Schlemm's canal under direct vision may assist in the solution of the surgical problem of chronic glaucoma."

W. S. REESE.

SIMPLE CHRONIC GLAUCOMA AND ITS CONNECTION WITH CONGENITAL ANOMALIES OF THE EYE. A. F. RUMIANTZEVA, Vestnik oftal. **11:** 348, 1937.

Rumiantzeva reports 5 cases of simple chronic glaucoma in persons between the ages of 20 and 35 with various congenital anomalies, such as heterochromia, aniridia, microcornea, coloboma of the iris and polycoria. The condition was bilateral as a rule. She believes that because of atrophy and defects in the iris there is a disturbance of the filtration in the angle of the anterior chamber, in Schlemm's canal or in the perivascular space of the vorticose veins due to the abnormal development of the eye in embryonic life. Since at the aforementioned ages

the sclera is firm, no hydrophthalmos develops. Early diagnosis and control of simple glaucoma are urged.

The literature on the subject is reviewed. O. SITCHEVSKA.

Injuries

REMOVAL OF LEAD SHOT FROM THE VITREOUS BY USE OF THE BIPLANE FLUOROSCOPE. W. E. BORLEY and E. LEEF, Am. J. Ophth. **20:** 1232 (Dec.) 1937.

Borley and Leef report a case of extraction of lead shot from the vitreous by a modification of Cross' method. They give the following conclusions:

"(1) It is possible to remove lead shot or other nonmagnetic intraocular foreign bodies with the help of the double-plane fluoroscope. (2) The serious complications of retinal detachment which frequently follow extraction of these foreign particles, may be prevented by application of diathermic microcoagulation. (3) Early operation is essential to the maintenance of good vision." W. S. REESE.

OPERATIVE CLOSURE OF PERFORATING WOUNDS OF THE CORNEA. E. HERTEL, Arch. f. Ophth. **139:** 1 (Aug.) 1938.

Of the two available methods for treating perforating wounds of the cornea, corneal suture or covering and bracing of the wound with a conjunctival flap, the former affords closer approximation and apposition of the lips of the wound. The author carries the corneal sutures through the anterior half of the cornea and through a previously prepared conjunctival flap. The sutures are tied on the anterior surface of this flap, damage to the corneal surface by the threads and knots thereby being avoided, and the flap is held in place. Numerous drawings of pathologic sections illustrate the various (histologic) modes of closure of corneal wounds. P. C. KRONFELD.

Lacrimal Apparatus

TREATMENT OF CONGENITAL ATRESIA OF THE NASOLACRIMAL DUCT. S. LARSSON, Acta ophth. **16:** 271, 1938.

The nasolacrimal duct may be longer than the bony canal containing it. In such cases the duct continues below the orifice of the bony canal, the medial wall of the duct being covered by nasal mucous membrane. The normal opening of the duct may form a window on the medial wall above the end of the duct. For this reason there are certain patients with congenital atresia of the lacrimal duct who cannot be successfully treated by probing, for the probe passes to the floor of the nasal fossa without producing a perforation medially. In 2 such instances the author has removed the tip of the inferior turbinate and incised the mucosa against a probe which had previously been introduced in the usual way.

O. P. PERKINS.

Lens

CATARACT ASSOCIATED WITH AN HEREDITARY RETINAL LESION IN RATS. M. C. BOURNE, D. ADAMS CAMPBELL and M. PYKE, Brit. J. Ophth. **22:** 608 (Oct.) 1938.

The animals studied were bred from cataractous rats which were otherwise healthy and did not suffer from dietary deficiency or traumatic injury. The cataracts appeared in successive generations. The authors were satisfied that some hereditary factor was concerned in their production.

Histologic examination showed that although the iris and ciliary body were normal, cataractous changes in the lens were invariably associated with a typical form of retinal degeneration.

The variations in the time of onset and in the rate of development suggested that the cataract is a secondary but not a "terminal complication" resulting from retinal disintegration. There were occasional persistent remnants of the hyaloid artery and fibrous strands in the vitreous.

Illustrations accompany the article. W. ZENTMAYER.

THE CHOLESTEROL CONTENT OF NORMAL AND OF CATARACTOUS HUMAN LENSES. E. BUNGE, Arch. f. Ophth. **139:** 50 (Aug.) 1938.

The cholesterol content of clear human lenses extracted post mortem was found to increase with age (colorimetric determination of the digitonin precipitate). At the age of 70 the lens contained about six times as much cholesterol as at the time of birth. Fifteen intra vitam (intracapsularly) extracted cataractous lenses were analyzed and found to contain essentially the same amounts of cholesterol as clear lenses of persons of the same age if the difference in water content between mature cataracts and clear lenses was taken into consideration.

P. C. KRONFELD.

Methods of Examination

QUANTITATIVE PERIMETRY IN GLAUCOMA WITH OBJECTS OF VARYING LUMINOSITY. J. ROLL, Klin. Monatsbl. f. Augenh. **100:** 600 (April) 1938.

Roll examined the field of vision of glaucomatous patients with objects of reduced luminosity, as provided on Zeiss' projection perimeter. Using these objects in the dark room, he found that defects in the field of vision, noticed under usual conditions, were more distinct and more extensive. No difference was observed in cases of recent glaucoma in which perimetric examination or Bjerrum's screen had revealed an absence of defects. In cases of more advanced glaucoma distinct contractions were noticed in the field for white and colors, after a normal outline was found with bright objects. This method allows definite conclusions regarding the activity of the periphery of the retina in chronic glaucoma. Roll expects to gain some information as to the therapy.

K. L. STOLL.

Neurology

OCULAR SIGNS OF INTRACRANIAL DISEASE IN CHILDREN AND JUVENILES. E. W. NEWMAN, Am. J. Ophth. **21**: 286 (March) 1938.

Newman studied 42 cases of intracranial disease in children and juvenile patients and presents the following summary:

"1. Changes in the optic discs were found in 95.2 per cent of cases; this fact emphasizes the importance of an ophthalmoscopic examination in all children with findings which might in any way lead to a suspicion of intracranial disease.

"2. Loss of vision was registered as a complaint in only 23.8 per cent of the cases in spite of the fact that 95.2 per cent of the cases presented abnormalities of the optic discs. This is due to the fact that children do not appreciate failure of vision so readily as do adults, and that choked discs do not affect the vision for some time.

"3. Nystagmus was present in 21.4 per cent of the patients, all of whom had tumor of the brain and, with one exception, occurred with a tumor of the cerebellum.

"4. Of the total number of patients in this series, 55 per cent had brain tumors. Histological verification was possible in all except two cases, in which the cerebellar lesion was seen at the time of decompression. The optic discs were normal in only one case, a subtentorial lesion.

"5. A case of high-cervical-cord tumor associated with bilateral choked discs is reported, and a short review of the literature pertaining thereto is cited.

"6. This study emphasizes the extreme importance of a thorough ocular examination. The omission of a fundus examination is often the reason for failure in reaching a correct diagnosis; this is especially true in the case of children, in whom other examinations may be difficult."

W. S. REESE.

REDUCTION OF INCREASED INTRACRANIAL PRESSURE BY CONCENTRATED SOLUTIONS OF HUMAN LYOPHILE SERUM. JOSEPH HUGHES, STUART MUDD and EDWARD A. STRECKER, Arch. Neurol. & Psychiat. **39**: 1277 (June) 1938. REDUCTION OF CEREBROSPINAL FLUID PRESSURE BY CONCENTRATED LYOPHILE SERUM. DAVID WRIGHT, DOUGLAS BOND and JOSEPH HUGHES, ibid. **39**: 1288 (June) 1938.

Because variations in the osmotic pressure of the blood influence the intraocular as well as the intracranial pressure, the use of concentrated lyophile serum may become important clinically for the reduction of intraocular as well as of intracranial pressure.

Hughes, Mudd and Strecker, using a preparation of four times the concentration of normal serum, report in 7 patients an increase in blood pressure and a decrease in cerebrospinal fluid pressure for

relatively long periods. In 3 normal subjects they found this serum more effective than sucrose.

Wright, Bond and Hughes found that concentrated solution of serum in doses of ·4 cc. per kilogram of body weight reduce cerebrospinal fluid pressure of dogs for relatively long periods, as indicated by continuous recording of cisternal pressure. Eight cubic centimeters of serum per kilogram of body weight maintained a reduction in pressure for longer than twenty hours.

The results of the animal experiments reported in the second paper confirm the clinical observations presented in the first paper.

R. IRVINE.

Ocular Muscles

A PHOTOGRAPHIC ANALYSIS OF ALTERNATING VISION DURING READING. B. CLARK, Am. J. Ophth. **20:** 1142 (Nov.) 1937.

Clark gives the following summary:

"The binocular behavior of a group of 10 university students was studied during control or bar-reading. All of the subjects showed normal exophoria at the reading distance. The results of this study lead to the following general conclusions:

"1. The alternating vision that occurs during bar-reading does not appreciably alter reading efficiency as measured by eye-movement photography.

"2. The bar-readers showed much more variation in binocular behavior than did a control group during normal reading. This variation was found both in convergence and divergence in reading through the lines, in spite of the fact that binocular vision was present for the last fixations at the ends of the lines."

W. S. REESE.

THE EFFECTS OF ANOXEMIA ON OCULAR MOVEMENTS WHILE READING. R. A. MCFARLAND, C. A. KNEHR and C. BERENS, Am. J. Ophth. **20:** 1204 (Dec.) 1937.

Following experiments on 20 patients, 8 of whom were controls, in a Barach oxygen chamber the following conclusions were made:

"1. A significant increase was found in the reading time of control and experimental patients in oxygen concentrations corresponding to 15,000 to 18,000 feet. A similar increase was found in the number of fixations. A significant increase in regressions was made by the experimental group and a slight decrease (not significant) by the control group.

"2. The visual axes of patients with normal refraction and muscle and those with diminished acuity, muscle imbalance, and heterotropias showed (1) divergent movements during reading fixations and (2) convergence during the saccadic movements in normal and reduced oxygen concentrations. Since this occurs even in esotropia it may be related to variations in vergence caused by changes in accommodative effort.

"3. A decrease in the extent of divergent movements occurred in reduced oxygen for both groups, and was significant for the controls at 18,000 feet.

"4. The data showed no statistical relationship to the eye examination for the two groups as a whole. When the photographic records were analyzed individually, however, significant alterations were observed which could be definitely related to the findings from the eye examinations.

"5. The general qualitative characteristics of the ocular movements for both groups showed a diminution of precision in oxygen want, with the appearance of nystagmoid movements, general unsteadiness, and accentuation of abnormalities.

"6. A decrease in comprehension of the reading material was observed for all of the patients in reduced oxygen. This may represent an impairment of perception or of memory for immediate recall.

"7. The data from 10 patients indicated a tendency toward acclimatization by the end of one hour at 13,000 feet (12.5 per cent O_2.). No such adjustment was detectable at 18,000 feet (10.5 per cent O_2.).

"8. Photographic records of ocular movements obtained during reading appear to be of some value in the diagnosis of clinical anomalies. This method objectively records the movements of the eyes in a normal situation. Under conditions of anoxemia the latent defects present become accentuated. Therefore, this procedure may be of clinical significance.

"9. The eye-movement photographs showed that reading time per line and adjustments during fixations were sensitive measures of the early effects of anoxemia.

"10. The decrease in efficiency of ocular movements under anoxemia may be attributed to the diminished amount of oxygen being delivered to the nervous tissue, subcortical as well as cortical." W. S. Reese.

Orbit, Eyeball and Accessory Sinuses

A Comparative Study of Experimental and Clinical Exophthalmos. G. K. Smelser, Am. J. Ophth. **20:** 1189 (Dec.) 1937.

Smelser reviews the theories of the cause of exophthalmos in exophthalmic goiter and some of the experimental work; he also reviews the clinical data, this consisting mainly of biopsies in 6 cases. Approximately 100 young adult guinea pigs were used. Of these, 50 were thyroidectomized, and one cervical sympathetic ganglion was removed from half of them. Both males and females were included, some of them being gonadectomized. They were fed normal stock diet. The extract used was prepared from acetone dried bovine anterior pituitary lobes from which the posterior lobes had been carefully removed. Thyroidectomy had no obvious effect on the eyes. Removal of the cervical sympathetic ganglion produced a definite ptosis, enophthalmos and a small pupil in the effected eye. Injection of the pituitary extract into normal guinea pigs produced no demonstrable effect, whereas in all but 3 of the thyroidectomized animals a definite and, in some instances, an extreme exophthalmos was induced, this usually appearing in from ten to twenty

days. After enumerating the histologic changes and discussing them, Smelser gives the following summary:

"Clinical

"1. Exophthalmos in the cases studied was due to excess orbital tissue.

"2. Retrobulbar connective tissue, fat, and muscles were edematous and infiltrated with wandering cells.

"3. Similar changes were found in hyper and hypothyroid cases.

"4. Extraocular muscles were enlarged, though muscle-fiber degeneration was not striking.

"Experimental

"1. Exophthalmos due to increased orbital tissue was produced in thyroidectomized guinea pigs.

"2. Retrobulbar connective tissue, fat, and muscles were infiltrated with edematous material indistinguishable from that in clinical cases.

"3. Exophthalmos persisted post mortem.

"4. Removal of the cervical sympathetic ganglion did not inhibit the increase in orbital tissues.

"5. Areas of round-cell infiltration were found in the fat and muscles."

W. S. Reese.

Abnormal Pneumatization of the Sphenoid Sinus and Optic Neuritis. L. Giacobbi, Riv. oto-neuro-oftal. 14:317 (May-June) 1937.

Giacobbi is of the opinion that roentgenographic examination of sinuses should be made in cases of optic neuritis not only to ascertain the presence of infection but to obtain the dimensions of the sphenoid sinus. He reports 3 cases of optic neuritis in which no infection could be found on rhinoscopic or roentgenographic examination. The sphenoid sinuses were unusually large, and Giacobbi is of the opinion that this was the factor responsible for the disease of the optic nerve. In such cases opening the sphenoid sinuses seems to have a definite beneficial effect.

F. P. Guida.

Physiologic Optics

Entoptic Images of the Pupil Seen by Double Reflection Through Eye Glasses. G. Sanna, Ann. di ottal. e clin. ocul. 66:282 (April) 1938.

The author attempts to explain a phenomenon noted by himself and others. When a person wearing glasses enters a room lit by an intense electric light, with the gaze directed to the less illuminated wall, a luminous disk is seen with either eye. It varies in size with the distance of the glasses from the eye and with the power of the convex lens employed, becoming larger as the power of the lens increases and smaller as the lens is removed from the eye. The author proves by optical

theory and experiment that the image is that of the pupil reflected first from the corneal surface and then from the posterior surface of the lens. If the lens is increased in power, a second smaller image, corresponding to reflection from its anterior surface, is seen.

S. R. GIFFORD.

Physiology

PRESSURE OF THE CENTRAL RETINAL ARTERY IN RELATION TO THE CAROTID SINUS REFLEX. C. GANDOLFI, Ann. di ottal. e clin. ocul. **66:** 132 (Feb.) 1938.

Compression of the carotid sinus at the level of the upper margin of the thyroid cartilage produces in man a slowing of the pulse. The opposite effect is produced by compression of the common carotid artery. The author noted the diastolic retinal pressure with Bailliart's dynamometer in 20 normal youths and young adults before and during compression of the sinus. The general blood pressure showed a rise of from 10 to 25 mm. during compression, and the diastolic retinal pressure rose from 5 to 20 mm. The rise in pressure on the side on which compression was made and on the opposite side was the same, a result which differs from Baurmann's findings. The effect is due to a vasomotor reflex.

S. R. GIFFORD.

DARK ADAPTATION IN SLIGHTLY PIGMENTED AND ALBINOTIC EYES: REPORT OF CASES. E. BUNGE and W. HEYN, Klin. Monatsbl. f. Augenh. **100:** 178 (Feb.) 1938.

The authors describe the technic and calculations used in their study of dark and light adaptation, which were devised in a manner to avoid errors. They examined 8 albinotic patients and a young man with normal pigmentation except for an albinotic fundus. The course of dark adaptation was recorded in graphs. The apparatus of Drescher and Trendelenburg was used for light adaptation, and the values were registered with an adaptometer devised by Engelking and Hartung. The values for the dark adaptation of normal eyes were found with the same method by examining the eyes of 20 normal persons. Patients with total albinism, who were examined repeatedly, showed the same figures for the beginning and final adaptation as normal persons. Ten light blond persons presented the same course and type of adaptation as 10 persons with dark hair.

K. L. STOLL.

Refraction and Accommodation

NEAR VISION IN MYOPIA. E. DI BARI, Arch. f. Ophth. **139:** 105 (Aug.) 1938.

Because of the larger images which the retina of the myopic eye receives, clear vision without correction is possible at distances from the eye which slightly exceed that of the punctum remotum. The author, a physicist, deals with this phenomenon mathematically.

P. C. KRONFELD.

Retina and Optic Nerve

THE INDIRECT TRAUMATIC OPTIC ATROPHIES. M. DAVIDSON, Am. J. Ophth. 21: 7 (Jan.) 1938.

Davidson makes the following conclusions in this rather comprehensive article on indirect traumatic atrophy of the optic nerve:

"1. The condition may properly be described as a syndrome involving a certain type of accident, namely, falls from heights, and an evolution with immediate blindness, abolition of the direct reaction to light, without fundus changes at first, and optic atrophy in from one to three weeks.

"2. It is the result of accidents, principally industrial, among workers engaged in the building trade, in which falls are likely, rather than of compound skull fractures or of war injuries.

"3. The bilateral cases and the chiasmal syndromes are probably a more common but easily overlooked condition; lues and other intracranial pathology are factors to be considered in the bilateral cases, principally.

"4. The field defects observed suggest a diffuseness of optic-nerve and chiasm lesions rather than isolated bundle defects; the relation between the foramen fracture and the optic-nerve lesions is apparently the same as the relation between skull fracture and brain injury in general, and is not determined by the site of fracture; contralateral involvement is not rare.

"5. The apex syndrome, that is, involvement of optic foramen and sphenoidal fissure simultaneously, is rare, and when present it is the sixth nerve that is most frequently involved.

"6. All unilateral optic atrophies, remaining so under long observation and following a head injury, even minor, are to be regarded as the result of the head injury regardless of X-ray findings or history of unconsciousness.

"7. In industry, in the absence of routine preemployment and periodic eye examinations, an early inquiry into the condition of the eyes after every head injury, irrespective of its mildness, is essential not only for diagnosis and prognosis, but for the elimination of medicolegal controversies and of prevention of miscarriage of justice."

W. S. REESE.

OCULOCARDIAC REFLEX OBSERVED AFTER AN OPERATION FOR DETACHMENT OF THE RETINA BY DIATHERMOCOAGULATION. P. MICHAND, Bull. Soc. d'opht. de Paris 50: 136 (March) 1938.

The patient, 20 years of age, had a detachment of the superior internal quadrant of the retina following an injury. There was no hematoma of the ocular region. The general condition and pulse were normal. Sixteen hours after operation the pulse had a normal rhythm but had dropped to between 40 and 44 pulsations per minute. The respiration was likewise slightly retarded. A retrobulbar hemorrhage was considered, although it could not properly be verified. An immediate injec-

tion of alcohol was made retrobulbarly, and the pulse rate returned to 60 per minute.

Other patients who were operated on for various conditions showed a slowing of the pulse rate. The latency of the reaction and its probable mode of transmission are commented on.

L. L. MAYER.

THORACOCERVICAL SYMPATHECTOMY IN THE TREATMENT OF RETINITIS PIGMENTOSA. A. CHIASSERINI and I. NEUSCHÜLER, Riv. oto-neuro-oftal. **14:** 251 (May-June) 1937.

Chiasserini and Neuschüler review the medical and surgical methods of treating retinitis pigmentosa that have been used up to the present. They offer cervical sympathectomy as another possibility for the purpose of altering the choroidal and retinal circulation.

Of the 15 cases reported, improvement occurred in 9, the condition in 5 was unchanged and that in 1 case became worse.

The method has the advantage that not only does it preserve the vision present but it allows for an increase of visual acuity and often of the visual field.

The inconveniences noted were the establishing of the Claude-Bernard-Horner syndrome, hyperemia of the conjunctiva and lowering of the intraocular tension immediately after the operation. With the passage of time all these signs disappeared. A detailed description of the surgical procedure is included.

F. P. GUIDA.

BILATERAL LESION OF THE MACULA CAUSED BY RADIATION OF LIGHTNING: REPORT OF A CASE. W. HANDMANN JR., Klin. Monatsbl. f. Augenh. **100:** 438 (March) 1938.

A girl, aged 17, had perfect vision until her eyes were temporarily blinded by lightning. The surface, pupils and media were normal. In each macula a flat, slightly yellowish infiltration was observed, more marked in the left than in the right; these areas were shaped like a geographic map and faintly spotted gray, measuring about 2.5 disk diameters. The disks were free from congestion, and the retinal vessels were normal in appearance but the macular reflexes were absent. Pictures of both fundi are included. Constitutional diseases were excluded by tests in point. Vision increased from 6/18 in the right eye and 6/36 in the left eye to 6/5 and 6/18, respectively, after three weeks' rest in a dark room. After four months vision was nearly 6/5 in each eye, but the central scotoma persisted to some extent.

K. L. STOLL.

THE TREATMENT OF RETINITIS PIGMENTOSA BY LAUBER'S METHOD. I. A. VASSERMAN, Vestnik oftal. **11:** 868, 1937.

Lauber established by clinical and experimental studies that there is a definite relation between the diastolic pressure in the retinal arteries and the intraocular pressure. The decrease of the intraocular tension is favorable for the diastolic pressure and the nutrition of the retina, while the increase of the intraocular tension without a corresponding

increase of the general blood pressure is unfavorable for the circulation of the retina, and an atrophy of the optic nerve or retinitis pigmentosa may result. In order to maintain the normal equilibrium between the diastolic retinal and intraocular pressure, Lauber recommends that the intraocular tension be reduced with the administration of pilocarpine hydrochloride or by cyclodialysis and that the blood pressure be increased by injections of caffein or strychnine.

Vasserman treated 11 patients (21 eyes) from 18 to 51 years of age who were suffering from retinitis pigmentosa, the period of observation being from three to seven months. The functional tests of the eye and measurements of the blood pressure were done before, during and at various times after the treatment. A 2 per cent solution of pilocarpine hydrochloride was instilled into the eye three days preceding the injections. One cubic centimeter of a solution of strychnine in a concentration of 1:1,000 was injected subcutaneously daily up to thirty injections; the pilocarpine hydrochloride was instilled simultaneously.

The intraocular tension and its daily variations were found to be higher in eyes with retinitis pigmentosa than in normal eyes; it was decreased under the influence of miotics, and its daily variation curve was more regular. The blood pressure was low in the majority of the patients. In those patients in whom it could be raised by the injections of strychnine, the therapeutic result was good. The near and distance vision was improved in nearly all patients, while the visual fields were improved in only 3. Recent infections responded to the treatment more readily than the infections which had been neglected.

Vasserman concludes that simultaneous reduction of the intraocular tension and the increase of the blood pressure give favorable results in the treatment of retinitis pigmentosa; further observations are necessary. It was easier to reduce the intraocular tension than to increase the blood pressure.

O. Sitchevska.

Trachoma

Pathologic Appearance of the Cornea in Trachoma. A. Santonastoso, Ann. di ottal. e clin. ocul. 66: 401 (June) 1938.

The diagnostic value of corneal changes in trachoma is discussed. The author carefully examined the corneas of many small children with trachoma. Extensive vascularization was found in the corneas of a great number, and in some of the children with mild trachoma vessels passed the limbus. Other children with advanced trachoma were seen, however, in whom there was complete absence of vascularization. On the other hand, vascularization was found in children with slight conjunctivitis without any of the other diagnostic signs of trachoma. The author believes some individual sensitivity of the tissues at the limbus, possibly of an allergic nature, is responsible for the differences observed. He concludes that vascularization of the cornea, while frequent, must not be considered conclusive evidence for or against trachoma but that the diagnosis must be based on other signs and especially on the course of the disease.

S. R. Gifford.

Tumors

A Clinical and Anatomopathologic Contribution to Tumors of the Optic Nerve. L. Venco, Ann. di ottal. e clin. ocul. **66:** 321 (May) 1938.

The author reviews the literature and reports a personal case. A boy of 12 years was first seen at the age of 6, when proptosis and loss of vision on the right side had been noticed for two months. Vision in the right eye was 4/50. There was swelling of the disk. Permission for operation was refused, and the patient was not seen till a year later. A tumor the size of a small orange protruded from the orbit, the collapsed and shrunken globe being just visible on its surface. At operation the mass was removed almost intact, a few remains being dissected out piecemeal. Sections showed a swelling of the optic nerve just behind the globe which merged into the tumor mass. This was composed of cells originating from the interstitial cells of the optic nerve but differing in staining properties from both glia and collagen. The tumor is classed as a neurinoma.

S. R. Gifford.

Fibroma of the Sclera. J. Schmidt, Arch. f. Ophth. **138:** 748 (July) 1938.

The author reports the case of a man aged 46 in whom scleritis developed and persisted for about a year, when retinal detachment occurred. The region of the detachment appeared dark on transillumination, and the eye was enucleated. Pathologic examination revealed foci of round cell infiltration in the uvea; a similar but more diffuse infiltration of the sclera at the posterior pole, and a fibroma which had originated from the sclera and, pushing the overlying retina and choroid inward, developed into a large mushroom-shaped intraocular tumor. The author classifies this tumor among the rare primary fibromas of the sclera and considers the inflammatory process to be secondary to the neoplastic one.

P. C. Kronfeld.

Society Transactions

Edited by W. L. Benedict

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

James W. White, M.D., *Chairman*

Nov. 21, 1938

Rudolf Aebli, M.D., *Secretary*

Recurrence of Ocular Hypertension Eighteen Years After an Elliot Operation: Report of a Case. Dr. Mark J. Schoenberg.

Ocular hypertension recurred in the left eye of a patient eighteen years after an Elliot operation. During this interval the visual acuity remained unchanged and the tension normal. Curves of the ocular tension and visual acuity and records of the visual fields for the entire period of observation were made. (These were projected on the screen.)

DISCUSSION

Dr. Arnold Knapp: I think that Dr. Schoenberg's observation is interesting, particularly if there was no change in the filtering scar and the attack of glaucoma responded to treatment with miotics only. I have a case in mind in which the filtering scar ceased to function after eleven years. It occurred to me that this could be due to a change which the patient seemed to be undergoing. There was manifest atrophy of the orbital contents, with sinking in of the eyeballs, such as one would expect in myxedema. The thought came to me that a change in the amount of aqueous filtration might be a possible factor in some of these cases. It is generally known that success in trephining depends on early restoration of the tension, following which a satisfactory bleb is obtained. If the tension is not restored promptly, the bleb is slow in forming and may be inadequate. I believe that in my patient, in whom there was a tendency to myxedema, there was a diminution in the aqueous secretion, so that the filtering scar collapsed and closed up.

Dr. Ernest F. Krug: I wish to ask Dr. Schoenberg whether during this attack the chamber was very shallow and whether there was any evidence of swelling of the lens.

Dr. Mark J. Schoenberg: In regard to Dr. Knapp's question, there was no obstruction of the trephine opening; one could clearly see it with the aid of the biomicroscope. I am inclined to believe that the increase of tension was of a "functional nature." The tension would not have yielded so readily to the use of pilocarpine if the trouble had been due to an organic obstruction. I have no doubt that there are several stages in the evolution of ocular hypertension in glaucoma. In

the early stages the increase is functional, and a decrease can be obtained by the administration of pilocarpine. In some cases, as in the one reported by me, the glaucoma may remain in this stage for a considerable length of time.

In reply to Dr. Krug's question, there was no swelling of the lens. Biomicroscopic examination would have revealed this. The anterior chamber was not shallow, and there was no diminution of vision. If there had been swelling of the lens, I do not see how it could disappear after the use of pilocarpine for a few days.

Treatment of Inclusion Conjunctivitis with Sulfanilamide: Report of a Case. Dr. Phillips Thygeson (by invitation).

In view of the curative effect of sulfanilamide in trachoma, it was decided to test the drug on the closely related disease, inclusion conjunctivitis. Accordingly, the drug was given to 2 Macacus rhesus monkeys in which the disease had been produced experimentally. A daily dose of ½ grain (0.032 Gm.) per pound of body weight resulted in complete healing of the lesion in two weeks. There was no change in the conjunctivitis in 2 untreated control animals during this period. The drug was then tested on an adult. The condition was of two weeks' duration, was unilateral and was characterized by severe follicular and papillary hypertrophy of the conjunctiva, most prominent in the conjunctiva of the lower lid. There were preauricular adenopathy, pseudoptosis, bulbar injection and edema of the limbus but no epithelial or other corneal changes. Cultures and scrapings showed no significant bacteria but moderate numbers of epithelial cell inclusion bodies. The patient was hospitalized and placed on a daily dose of 30 grains (0.195 Gm.) of sulfanilamide. No local treatment was employed. Improvement was first noted on the second day, and after thirteen days the eye had returned to normal. Inclusion bodies, which averaged from 15 to 25 per slide at the onset of treatment, could not be found after the second day. The patient has been followed for three months after healing with no evidence of recurrence. In none of 51 previously observed patients (9 adults and 42 infants) with inclusion conjunctivitis who were followed throughout their clinical course did healing occur in less than three months.

DISCUSSION

Dr. Arthur J. Bedell: I can confirm the observations made by Dr. Thygeson. His photographic proof has been duplicated by me. The results following the administration of sulfanilamide are so remarkable that they are almost unbelievable.

I have been using a preparation which combines sulfanilamide with sodium bicarbonate. This is much more concentrated than the usual drug and will soon be on the market.

I was glad to see Dr. Thygeson's pictures, for they speak well for his results. I urge all physicians to try this treatment in properly selected and controlled cases.

Dr. Walter I. Lillie: My experience is similar to that of Dr. Thygeson. I have used the drug only since summer. The first case in which it was employed was very interesting. The patient, a woman

about 54 years of age who had subacute trachoma, on being told that she should discontinue all local measures and take treatment orally, said: "I have been treated in the best clinics in the East for the last eight years, and you are the first physician who has said 'you must discontinue all treatment about the eyes and take the medicine by mouth.'" "Well, madam," I said, "you have given the best clinics in the East a fair trial for eight years and you have not been cured, so if you care to gamble for two weeks you might try this method." She agreed to give the treatment a trial. On the fifth day she telephoned and said: "Doctor, my eyes have felt better the last two days than they have for eight years." When she returned to the clinic, practically all signs of trachoma had disappeared. At the present time she has only a few small active vessels, which appear as a pannus in the upper part of the cornea; the rest of the cornea is clear.

The dose I use is a little different from that used by Dr. Thygeson. For the first week I prescribe a third of the body weight in grains per day in conjunction with the same amount of sodium bicarbonate, and for the second week, a fourth of the body weight in grains. Usually two weeks' treatment more than suffices to eliminate all the acute inflammatory signs. It makes me wonder whether trachoma, as it is seen clinically, is a secondary manifestation of a streptococcic infection, since the streptococcus responds so specifically to sulfanilamide.

Dr. Richard Townley Paton: I was much interested in Dr. Thygeson's report, especially since a year and a half ago I began treating some patients with trachoma and severe conjunctivitis. The diagnosis at that time was uncertain, but the ocular condition was becoming progressively worse. I gave numerous subconjunctival injections of a 2.5 per cent solution of prontosil (disodium-4-sulfamidophenyl-2′-azo-7′-acetylamino-1′-hydroxynaphthalene-3′,6′-disulfonate), and I am not yet prepared to say that the method is without danger. I am extremely careful in giving the injections, as there is some danger of damaging the retina. I am favorably impressed with the results obtained in about 15 cases. I wish Dr. Thygeson would tell something about the subconjunctival use of sulfanilamide.

Dr. Phillips Thygeson: I have had no experience with the subconjunctival use of sulfanilamide, so I can offer nothing in this connection.

In regard to Dr. Lillie's suggestion of the possibility of the drug acting on a secondary bacterial infection in trachoma, I must say that the sulfanilamide apparently acts directly on the virus. The disease becomes noninfectious for baboons rapidly, which would not be the case if only a secondary bacterial agent were being acted on. Furthermore, the drug causes rapid disappearance of the epithelial cell inclusion bodies, also an indication of direct action on the virus.

Treatment of Diseases of the Eye with Grenz Rays. Dr. Raymond Pfeiffer.

This article will appear in full, with discussion, in a later issue of the Archives.

THE CLINICAL SIGNIFICANCE OF RETINAL CHANGES IN ARTERIAL HYPERTENSION. DR. WALTER I. LILLIE, Philadelphia (by invitation).

Arterial hypertension is a result of some change in the peripheral arteriolar bed which may be functional or organic. The retinal arterioles are the most accessible portion of the peripheral arteriolar bed for observation and study, and ophthalmologists have assumed an important role in aiding the internist to classify arterial hypertension properly by correctly interpreting the associated retinal changes.

These primary changes are observed in the retinal arterioles and may be functional or organic. The functional changes are those that may and do precede permanent organic damage. The rapidity of the hypertensive changes depends on how readily the peripheral arteriolar system responds or compensates to whatever is producing the clinical picture of hypertension. In essential hypertension, if the onset is not too precipitous, the retinal arterioles reveal a generalized constriction or attenuation without any irregularities in the lumen.

If the onset of the hypertension is precipitous, the functional changes observed in the retinal arterioles develop more rapidly and are more severe but may be transitory. Superimposed on the general attenuation or constriction of the arterioles are localized spasms of the arterioles, which in turn, depending on the severity of the spasm, produce an associated localized ischemia of the retina. These changes may be of extremely short duration, and the localized spasm or complete obliteration of the retinal arterioles should not be interpreted as a definite organic change at this stage.

Persistence of the general attenuation or constriction of the arterioles results in damage to the intima, which is revealed as irregularities in the lumen. When this occurs, sclerosis is now superimposed on the already described functional picture, which signifies permanent damage to the peripheral arteriolar system. When generalized attenuation of the arterioles is observed with evidence of sclerosis, graded from I to IV, with the associated widening of the arteriolar reflex stripe and arteriovenous compression, the patient can clinically be classified as having essential or benign hypertension. Associated with these retinal changes, a thrombosis of a branch of the central retinal vein sometimes occurs. This is purely a local change due to excessive compression by a sclerosed arteriole at its venous crossing and should not be confused with the retinitis of hypertension. The hemorrhages and exudates will absorb as soon as a collateral circulation is developed.

The retinal changes of severe hypertension are characterized by the presence of exudates and hemorrhages associated with mild angiospasm of the retinal arterioles, with irregularity of the lumen (sclerosis), widening of the arteriolar reflex stripe and arteriovenous compression but without edema of the disk. The presence of this type of retinitis is significant of severe vascular damage, and both the systolic and the diastolic pressure will be consistently higher than in essential hypertension.

Angiospastic hypertension may be divided into a preorganic and an organic phase. The term angiospasm signifies a narrowing of an arteriole, either partially or completely, to such an extent that it appears

as an ill defined line. When the peripheral arterial bed is precipitously affected without previous organic damage, the fundus presents a picture of edema of the disk and retina with associated cotton wool exudates and hemorrhages, with markedly attenuated and even completely obliterated arterioles but without evidence of sclerosis. Just how long angiospasm can persist without producing permanent damage to the arterioles is not known. If the basic cause can be eliminated before a permanent vascular change takes place, this type of retinitis can improve or may subside without residual vascular damage.

If the precipitous angiospasm is severe enough to produce early vascular damage, or if it is superimposed on a previously damaged peripheral arteriolar bed, the retinitis is characterized by edema of the disk and retina, with associated cotton wool exudates and hemorrhages and definite sclerosis of the retinal arterioles. When this change is observed, the retinitis of malignant hypertension is present. The presence of an incomplete or complete macular star, not infrequently seen in both preorganic and organic angiospastic retinitis, signifies that the retinal edema is absorbing and that the retinitis has been more severe. It does not mean that there is an associated nephritis, as often the renal function is adequate.

The acute retinitis which occurs somewhat precipitously as a terminal complication of glomerulonephritis is attributable to severe secondary anemia which accompanies the renal insufficiency. The disk, retina and choroid are anemic, and the edema, cotton wool exudates and hemorrhages are associated with normal retinal vessels. At times a diffuse edema of the retina accompanies the generalized edema of the body in cases of subacute or chronic glomerulonephritis. This type of retinitis is never seen in essential hypertension.

JAMES W. WHITE, M.D., *Chairman*

Dec. 19, 1938

RUDOLF AEBLI, M.D., *Secretary*

A NEW BINOCULAR GONIOSCOPIC APPARATUS. DR. DONALD BOGART, (by invitation).

The binocular slit lamp microscope has long fascinated inventive-minded ophthalmologists as a gonioscopic possibility. Suitable illumination, a support for the head of the microscope and a contact glass fulfil the requirements for a binocular gonioscope. Therefore, many instruments have been devised, the variable items being the support for the bulky head of the microscope and a source of light. The majority of instruments of this type possess the following disadvantages:

1. They are unwieldy, and the patient is in constant danger of being hit should any of the joints become loose. Most of the instruments hold the head in an upright, unbalanced position.

2. Many operators find it necessary to hold the head in their hands; therefore, high magnification is impossible. The instability of many

upright types of instruments give them but little advantage over the arrangement which is held in the hand.

3. The instruments are all costly.

4. The average busy physician does not have enough time in the routine of his practice to set up the usual bulky devices.

I have, with the help of Mr. Mangold, of Clairmont and Nichols, constructed a satisfactory gonioscopic apparatus in which the microscope is suspended instead of being held upright. Universal rotation and high degrees of magnification are possible. Goniotomy and goniophotography may be practiced, and the patient is in considerably less danger of being hit by the head of the microscope.

The support is made from a photographic copying stand with baseboard, upright and sliding arm, of the type sold by E. Leitz, makers of the Leica camera. The head of the slit lamp is removed, and the bottom joint is drilled to receive the heavy Leica ball and socket tripod head. This in turn is screwed into a small brass rod made to receive it; the rod is held in the end of the aforementioned sliding arm. The head of any standard slit lamp may be used.

Illumination is provided by the lamp used by Dr. Barkan. This is screwed to a rod, which is fitted to any type of head to be used. The lamp is usable on either alternating or direct current and keeps very cool. Any conceivable angle is possible between the objectives and the lamp, which burns at a rated 500 foot candles at its focus. The little lamp may be used as a pocket slit lamp or a transilluminator (suitable heads are available), thereby making up for the fact that its cost of $25 makes it the most costly item in the setup.

The quickly assembled apparatus is set up on a table or chair, with the patient reclining. A pillow is placed on the baseboard, and the ball and socket are centered over the eye to be examined. The entire angle may be explored by the examiner with the ball and socket loose, after the height and angle of the instrument are set. A turn of the setscrew anchors it firmly at any point, and higher power is possible by the substitution of another objective. The patient, whose head acts partially as a counter weight on the baseboard, is cautioned against getting up until the apparatus is steadied. I feel that the setup has the following advantages:

1. The cost is comparatively low. From $45 to $50 will buy the parts.

2. The apparatus is flexible, yet safe and stable enough for high magnification.

3. Illumination is adequate. The lamp may be used as a pocket slit lamp, a transilluminator or a general source of light.

4. The entire setup is easily portable.

5. The interchangeability of parts makes it possible for the owner of a Leica or Contax camera to use the copying stand for the copying of specimens or photographs or for actual photography of the eye in color or black and white, provided he gets the ground glass focusing attachment.

It is to be noted that the cost of parts mentioned does not include the slit lamp head or the contact glass.

I feel that the aforementioned reasons are sufficient excuse for presenting another gonioscopic apparatus. There would be no need for such an instrument (except for goniophotography and for extremely high power magnification) were it possible to obtain an instrument such as that of Dr. Troncoso. The low cost and interchangeability of the parts of the apparatus described make it possible for every ophthalmologist who has access to the head of a binocular slit lamp to possess a satisfactory gonioscope with little embarrassment to his budget or his time.

DISCUSSION

Dr. Manuel Uribe Troncoso: I am glad to hear that the method of gonioscopy is receiving more and more attention and that the medical profession is interested in the technic and the results of gonioscopic examination of the eye. To be practical to the clinician, examination of the angle of the anterior chamber has to be made with an instrument that everybody can use. As with the ophthalmoscope, one has to have a gonioscope which is handy, which can be used easily all around the angle and which does not cost too much. When one uses a complicated instrument in which a lot of machinery must be adjusted, a great deal of time is wasted and the contact glass is kept above the eye too long. The contact glass, especially the new, more convex model, is so heavy that it cannot be used for a long time on pathologic eyes. It is alright for normal eyes; a patient can tolerate examination with it for from ten to fifteen minutes, but not if he has glaucoma. In cases of glaucoma associated with hypertension and congestion of the eyeball it is necessary to make the examination quickly to avoid an increase in vascularity.

The objection to all the methods of examination with the slit lamp microscope is that the microscope has to be supported by the hands. As it is too heavy to be supported in this manner during a complete, prolonged observation all around the angle, a stand has to be used. Dr. Barkan, Dr. Hartshorne and, now, Dr. Bogart have devised special stands. The one used by Dr. Barkan which hangs from the ceiling is extremely complicated. The floor stand used by him is similar to the tripod used by Drs. Hartshorne and Castroviejo. Dr. Bogart has inserted a horizontal board under the mattress to hold the supporting shaft. This complicated machinery is alright for purposes of demonstration and for photography of the angle in the normal eye with a deep anterior chamber. However, when the chamber is shallow, especially in pathologic conditions, a convenient manual instrument in which the plane of observation can be changed easily from the surface of the iris to the recess of the anterior chamber is much better and gives more information. Besides, with the slit lamp microscope the illumination system, such as is used in Dr. Bogart's stand, does not follow the movements of the microscope, and the help of an assistant is necessary to focus the beam properly. My own gonioscope, which has a periscopic arrangement, can be turned easily around the center of the eye, and the examination is made quickly. Unfortunately the presence of the prisms makes the focusing of the instrument a little difficult. That is why the instrument has not been popular. I hope in the near future to have another gonioscope made with which one can have direct vision, so that the student and the practitioner may have the advantage of easy

manipulation without prisms. Still, I consider the prism important when the angle is very narrow and I want to see if the recess of the angle is closed or not. With the new contact glass and the gonioscope, one examines the side of the angle close to the observer, not the opposite side. I think that one can keep the instrument on a stand, such as Dr. Hartshorne's or Dr. Bogart's or my own, for purposes of demonstration but that one should have a handy instrument which has good magnification, gives a strong, steady illumination at a proper angle and easily shows the angle all around the eye.

Congenital Type of Endothelial Dystrophy. Dr. Frederick H. Theodore (by invitation).

This article appears in full in this issue of the Archives, page 626.

Atypical Primary Degeneration of the Retina (Retinitis Pigmentosa Sine Pigmento): Report of Occurrence in Three Members of a Family of Five. Dr. Samuel P. Oast.

Three members of a family of 5 children (all adults) began having night blindness in adolescence. Twins, now aged 27, have so far escaped. The fundi of all 3 are remarkably free from signs of pathologic changes to account for the profound visual disturbance, though some slight evidence of migration of pigment and retinal atrophy are present together with widespread distribution of small whitish dots in the retina, which brings up the subject of retinitis punctata albescens. The diagnosis of retinitis pigmentosa sine pigmento is made by studies of the visual fields which show the characteristic annular scotomas and peripheral depression of classic retinitis pigmentosa.

DISCUSSION

Dr. Arthur J. Bedell: I shall present four fundus photographs in color. Three are of patients suffering from retinitis pigmentosa; in each the zone of pigmentation was remote from the disk and could easily have been overlooked by a hasty observer.

In one patient the disk was of normal color and the retinal vessels were unchanged. In the second, the disk was slightly pallid, both arteries and veins were reduced in caliber, and in the macular region there were small diffuse depigmented areas; 4 disk diameters from the disk a typical zone of deposits the shape of bone corpuscles were seen. In the third, the disk was of a waxy color; the arteries and veins were decidedly contracted, and the pigmentation was marked. In all 3 night blindness was the symptom which led to the investigation.

The fourth picture is the fundus of a young myope whose chief complaint was night blindness, although the fundus showed no pigmentation.

The cases which Dr. Oast presented were of considerable interest, for the patients were examined at a stage when pigmentation was beginning. In all probability a few years from now they will show a decided change. Dr. Oast has performed a real service by reporting these cases, because they will lead to the earlier recognition and diagnosis of retinitis pigmentosa before pigmentation.

CHORIORETINITIS OF THE MACULAR AREA. DR. RAYMOND EMORY MEEK.

When I was requested to give a talk on the diseases of the choroid several years ago, I was extremely bored. I thought that there could be no more uninteresting section of the eye.

I had known that the choroid was a blood vessel coat composed of five layers, the lamina vitrea, or Bruch's membrane, the chorio-capillaris, the layer of small blood vessels, the layer of large blood vessels and the lamina fusca, but this seemed all there was to know. In studying the choroid, it proved to be very interesting.

The choroid is the layer which supplies a great part of the nourishment to the interior of the eye. Toxins may readily pass through Bruch's membrane and cause irritation of the retina. It is difficult for cellular exudate and micro-organisms to pass through the membrane and actively attack the retina. It may, however, become permeated with exudate if the inflammation is severe.

Some authorities explain the increase in tension as being a congestion of the choroid with a corresponding increase in volume. Irritation and congestion of the choroidal vessels through stimulation of the sympathetic nervous system may bring this about. The choroid is said to be erectile tissue and to expand readily when congested. This explains why the angle of the anterior chamber is obliterated when a patient has an attack of glaucoma; the sclera, being inelastic, will not give way, and the increased volume inside the eye causes the lens and iris to be pushed forward. This blocks all egress of the fluids of the anterior half of the eye. The attack may cease as rapidly as it began. The shrinking of the choroid allows the lens and iris to sink back, opening the angle and permitting the outflow of the aqueous in the anterior chamber. During repeated attacks, the vessels lose their tone. Thus the choroid remains permanently engorged, and chronic glaucoma is established.

The choroid is necessary to the nourishment of the outer half of the retina; in cases of detachment of the retina the retinal system of vessels may nourish the whole retina for a long time, but slowly the outer half of the retina dies. Hence, the earlier a detached retina is operated on and its double supply of nourishment restored, the more successful the operation is in establishing function.

The choroid is less elastic than the retina, and so in cases of severe contusions of the eye the choroid is ruptured readily and the retina remains intact over it, although the retina may also rupture. Small excrescences or verrucae often develop on Bruch's membrane; these appear as small white dots in the fundus. They are frequently seen in the aged and are spoken of as drusen.

In cases of severe choroiditis the choroid is often destroyed without the retina showing much change. The layer of retinal pigment is dissolved except at the margin of the inflamed area, where it is irritated and proliferates, giving rise to rings of pigment surrounding the choroidal lesion.

In cases of acute miliary tuberculosis the large tubercles may be seen scattered through the choroid; usually three or four are observed,

although as many as seventy-three have been noted. They appear under the retina as large white patches composed of giant cells, and in some areas caseation develops. The surrounding choroid shows little irritation. The retina usually is not involved.

The mesodermal origin of the choroid accounts for its being the seat of development of sarcomas.

I am reporting the case of a patient whom I have observed for about eleven years. When she first consulted me in 1927 she was 36 years of age, well nourished and apparently in good health, but on examining her fundi I found a patch of old healed choroiditis in each eye above and temporal to the disk. At that time a thorough check up revealed nothing of significance but a slightly positive tuberculin reaction. Injections of tuberculin were given, and from time to time since then I have examined the patient but have found no change in the fundus picture. Vision had remained 20/15 in each eye.

On Nov. 6, 1938, the patient consulted me because the vision had been poor in the left eye for two or three weeks. On examination I found the vision to be 20/15 in the right eye, while that in the left eye was limited to ability to distinguish fingers at 4 feet (122 cm.).

The fundus in the right eye showed no change in the patch of choroiditis, but there seemed to be slight edema of the retina over the temporal side and some little white flecks which might be exudate scattered through the macular area.

In the left eye there was an active process around the patch of choroiditis with a new process beginning above it. There was more marked edema of the temporal portion of the retina, and flecks which appeared to be exudate were scattered throughout the macular area.

The patient was put to bed and fever therapy instituted. There was no improvement. Next, sulfanilamide was tried but was discontinued when bile salts appeared in the urine.

A roentgenogram of the sinuses was normal, and a provocative Wassermann test and other tests proved negative, with the exception of the Mantoux test, which gave a 2 plus reaction.

My conclusion was that the condition consisted of a lighting up of an old tuberculous process, and I began injections of tuberculin again. However, rest in bed and the injections were without benefit.

As the patient began to complain of some nasal disturbance, I had Dr. Frank C. Carr see her, although the roentgenograms of the sinuses were normal.

His examination revealed polypoid growths in the nostril with pus coming from the right sphenoid and ethmoid sinuses. He advised immediate operation, which he did on December 2. Within eleven days the vision had returned to 20/200, and the edema appeared to be less in the retina. The patient was feeling better in every way. In fourteen days the vision was 20/50.

The purpose in presenting this case is to show the importance of checking the sinuses and then double checking them. Also, it is too easy to be misguided by a positive tuberculin reaction. Furthermore, an old lesion may mask symptoms and make one think that there is a lighting up of an old process rather than a new infection.

BACTERIAL SYNERGISM IN NECROSIS OF THE LID: REPORT OF A CASE. DR. ISADORE GIVNER.

A man, aged 39, presented himself with a history of an acute infectious process involving the temporal portion of both lower lids ten months previously. At the time of the examination over one third of the outer portion of the lower lid of each eye seemed eaten away, the loss including the cilia, skin, tarsus and mucous membrane. The ulcerated infected area was cleansed, and a culture taken showed a hemolytic Staphylococcus aureus and a facultative aerobic Streptococcus viridans.

Experimentation done with the cultures brought out the observation that 1 cc. of a pure culture of either organism injected alone into the skin of a rabbit produced no reaction, whereas one-half the quantity of each injected together produced marked necrosis of the skin and underlying tissue.

Reconstruction of the lid was finally done by Dr. W. Hughes after his method of reconstruction on the lower lid, and an excellent result ensued.

THE FUNDUS EVIDENCE OF OCULAR INJURY. DR. ARTHUR J. BEDELL, Albany, N. Y.

Eighty colored photographs of fundi will be presented as the basis for the demonstration of lesions of the fundus following trauma. Perforations of the eyeball and intraocular foreign bodies are excluded; only the visible signs of injury to the fundus are presented. The prognosis in any accident should be withheld until the details of the fundus become visible.

DISCUSSION

DR. MORRIS DAVIDSON: These pictures may mislead one and give a wrong idea as to the extent of changes in the fundus from injuries. They are only photographs of injuries at the posterior pole. Most injuries, however, are in the periphery of the fundus. Such injuries, of course, are not accessible to photography.

I should like to challenge Dr. Bedell's unjustified statement about workmen's compensation. He does not deal with so-called compensation courts alone; he also deals with state ophthalmologists in the courts, who are impartial and who protect equally the claimant's and the insurance carrier's interests. No, Dr. Bedell does not deal solely with compensation courts.

DR. ARTHUR J. BEDELL: I disagree with the statement last made. I believe that when one goes before a referee who is legally appointed to receive evidence that one is to all intents and purposes in a court. I am afraid that Dr. Davidson would like to make me believe that I, as a witness, should agree or disagree with the physicians employed by the state. My function is to present what I believe is true evidence and to make deductions based on the facts.

Dr. Davidson says that most of the lesions following trauma are in the periphery of the fundus. If they cannot be seen through the photographing ophthalmoscope, the camera, and few are examined pathologically, on what can he base his assertion?

Dr. Morris Davidson: I did not say that the majority of these lesions are in the periphery of the fundus as the result of guess work. I am speaking of lesions in the periphery of the fundus which are visible on indirect ophthalmoscopic examination. They are not often visible on direct ophthalmoscopic examination. Furthermore, these lesions in the periphery of the fundus are part of the well known traumatic syndrome of the anterior segment, in which the nature of the lesions in the fundus is vouched for by accompanying lesions in the iris, lesions in the equator of the lens, i. e., opacities and pigment in the vitreous, and chorioretinal lesions, i. e., pigmented scars in the extreme periphery of the fundus, very often close to the ora serrata, which are visible on indirect ophthalmoscopic examination; so that this is not guess work. The lesions are actually there. In addition, there are partial scleral ruptures which might be described as giving rise to lesions in the periphery of the fundus which are likely to be missed but which can be seen and verified by examining the sclera.

Dr. Arthur J. Bedell: The only thing to be gotten from Dr. Davidson's reply is that there is pigmentation at the periphery. Surely this is not a new fact, for I have shown slides demonstrating it at this meeting.

Dr. Morris Davidson: I wish Dr. Bedell would study such lesions and attempt to photograph them.

Dr. Arthur J. Bedell: I do not think Dr. Davidson is justified in stating that the majority of traumatic injuries to the fundus are outside the posterior pole. I do not think that is borne out by the experience of most ophthalmologists.

Dr. Morris Davidson: It is borne out by a study of mine reported under the title of "The Minor Sequelae of Eye Contusions." I recorded the incidence of lesions of the posterior pole and the incidence of lesions of the peripheral portion of the fundus, and the latter amounted to about 58 per cent.

Dr. Arthur J. Bedell: I am sure that Dr. Davidson and I could carry this on endlessly without benefit to the listeners.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

Edward Stieren, M.D., *President*

Jan. 23, 1939

George H. Schuman, M.D., *Secretary*

Louis W. Statti, M.D., *Reporter*

Gliomatosis of the Optic Nerve: Report of a Case. Dr. Robert J. Billings.

Mrs. J. T., aged 48, an Italian housewife, a brunette, was first seen on Oct. 30, 1937, complaining of blindness in the left eye. There was a

history of an accidental blow five years before but with no immediate symptoms or effects. Two years ago she noticed dimness of vision in the eye and the appearance of black spots before it, which increased in number and gradually formed into a dark black mass. There was no history of hereditary disease. She had had high blood pressure for the past several years (193 systolic and 110 diastolic). The Wassermann reaction was negative. The general physical examination otherwise gave negative results, and the laboratory findings were essentially negative.

The vision of the right eye was 6/9, corrected to 6/6 with glasses, and the eye showed no abnormal changes externally or internally.

The left eye could perceive only hand movements at the extreme temporal periphery of the visual field. The cornea was clear; the iris was heavily pigmented, brown and healthy in appearance, and the pupil was 5 mm. in diameter. It reacted sluggishly to light and dilated freely but unevenly under cycloplegia with homatropine hydrobromide. Back of the lens there was an opaque brownish mass, about 5 by 8 by 12 mm., lying in the anterior portion of the vitreous chamber, with its flat side against the temporal wall of the globe. Deep oblique illumination of the angle showed a flat bright red mass back of the lens, which was believed to be a recent hemorrhage. No fundus details were visible on ophthalmoscopic examination. The slit lamp revealed a furry gelatinous surface to the mass and many red blood cells in the anterior vitreous. Transillumination and Wheeler's sign were positive for a globe impervious to light except for a narrow superior zone. The intraocular tension was 30 mm. (Schiötz). A provisional diagnosis of sarcoma was made, and the case was presented before the Pittsburgh Ophthalmological Society on Nov. 23, 1937, where it aroused considerable discussion. Enucleation was deemed advisable because of the possibility of a malignant growth, and was performed on Dec. 3, 1937, with the patient under a general anesthesia. Convalescence was uneventful.

The diagnosis based on macroscopic and microscopic examination of the specimen was: a large, organized hemorrhage in the vitreous; a smaller, more recent hemorrhage in the vitreous; retinal arteriosclerosis; degeneration of the retinal ganglion cells; melanosis, and, most surprisingly, gliomatosis of the optic nerve.

Exfoliation of the Anterior Capsule of the Lens. Dr. Jay G. Linn.

Two women, both over 60 years of age, presented a senile flaking of the anterior capsule of the lens in both eyes. In each case no other pathologic change was noted except a slight elevation of tension in one and vascular sclerosis in the other. Dr. Stieren pointed out the close relationship of exfoliatio lentis to glaucoma by calling attention to the following statistics presented by Horven in 1936: Of 43 patients with flaking, 40 had glaucoma, and of 150 patients with glaucoma, 128 had flaking. Trauma to the iris as a causative factor was brought out when it was noted that in a similar case exfoliation of the capsule of the lens did not occur at the point where iridectomy had been performed. The use of miotics was suggested to immobilize the iris and prevent rubbing of the anterior capsule.

LOSS OF VITREOUS. DR. EDWARD A. WEISSER.

A man, aged 39, had his left eye enucleated in 1933 for phthisis bulbi following trauma. In 1937 his right eye presented a mature cataract, and extraction was advised. At completion of the corneal section there was an immediate and rapid flow of liquid vitreous. The speculum was quickly removed and the eye closed for about five minutes. At the end of this time, with the lids held open by retractors, iridectomy was performed and the lens delivered with the aid of a wire loop, with slightly more loss of vitreous. Sterile warm physiologic solution of sodium chloride was injected into the globe with a pipet. No attempt was made to replace the pillars of the iris. A binocular bandage was applied, and the usual postoperative management was carried out. At the first dressing there was little postoperative injection; the anterior chamber was fairly well reformed, and the corneal wound, flap and pillars of the iris were in good position. The patient left the hospital at the end of two weeks with vision of 6/60. He returned home to the northern part of the state to the care of the referring ophthalmologist. Recently this ophthalmologist reported that the patient sees fairly well and is able to do odd jobs about the house and to go out alone.

ANIRIDIA. DR. CHARLES KUTCHER.

Aniridia occurred in 3 members of the same family, the mother, aged 47; a son, aged 7, and a daughter, aged 9. There was no history of consanguinity, nor were any other members similarly afflicted. All 3 persons had posterior cortical cataracts; in addition, the mother had microphthalmos of one eye, and 1 of the children had corneal opacities. Two other daughters in the same family had normal eyes.

RETRACTION SYNDROME. DR. JOHN S. PLUMER.

A man presented a typical Duane retraction syndrome in the right eye, which as a rule occurs in the female sex. Uncorrected vision in the right eye was 6/15 and in the left eye, 6/7.5. The ocular fundi show no pathologic changes.

RETINAL DETACHMENT. DR. LOUIS W. STATTI.

E. S., aged 16, was struck in the right eye by a mushball in January 1937. Since then he had had a gradual decrease in vision. Examination six months later revealed a large retinal detachment, which included the entire lower half of the retina and the macular area. There was a large triangular retinal tear at 5 o'clock in the periphery, with the base extending toward the ciliary body. Vision was limited to perception of hand movements, and there was a corresponding defect in the visual field. The left eye was normal.

On June 9, 1937, electrocoagulation was carried out. The sclera was exposed over the entire lower half of the globe; the muscles were not resected. A single point needle about 2 mm. in length was used, and after the area above the tear was encircled with single punctures, extending through the sclera and the choroid, the remainder of the exposed sclera was punctured about fifteen times, several points reaching back as far as the equator. A mixed cutting and coagulating current

was used. The operation was repeated about two weeks later; at this time the punctures were localized over the lower nasal quadrant, owing to the fact that the retina was still detached at this point. Recovery was uneventful.

One month after operation vision in the right eye was 20/70, and the field was normal. At the time of this report vision is 20/30, and the retina is completely reattached.

Book Reviews

Anatomie der Sehrinde (Monographie aus dem Gesamtgebiet der Neurologie und Psychiatrie, no. 64). By Prof. Max de Crinis. Price, 7.80 marks. Pp. 37, with 19 illustrations. Berlin: Julius Springer, 1938.

By a special staining method de Crinis has succeeded in demonstrating the ganglion cell bodies and the dendrites together, as with Golgi's method. He believes that the development of the dendrites expresses the higher or the lower functional value of a ganglion cell. He shows this in the visual cortex (as was done a few years ago in the auditory cortex) and gives a brief survey of the anatomy of this part of the brain. He describes the well known features about the extension of the cortical visual field in man and in animals. In the lower forms it is lateral, while in the higher forms it is located more medially. The extension of this visual field from the calcarine fissure to the lateral surface of the brain in man characterizes a higher type with a special development of the visual sense (see the brain of the painter Menzel). An accurate description of the cytoarchitecture, myeloarchitecture and angioarchitecture confirms for the most part the findings of Brodmann and of von Economo and Koskinas.

The author denies the existence of the coniocortex (powder-like cortex) of von Economo and Koskinas, because he found many dendrites on the cells in area 17, or Oc (calcarina). He could also distinguish, like Henschen, light and color sense cells. He agrees with Kleist that in the fourth layer of the calcarina there is a correspondence of the inner part with the contralateral region of the outer part and with the homolateral fibers of the optic radiation. The intermediate layer may link together the impressions of both fields. He describes in the same manner the other two areas (18 and 19, or Ob and Oa of von Economo and Koskinas), the parastriatal and peristriatal areas, with the disappearance of the special visual cells. The primary function of light and color vision is located in the calcarina. The so-called visuopsychic functions (mnestic, associative or opticomotor) are located in the parastriatal and peristriatal fields. These develop later than the calcarina, according to the dendrites (theory of cytodendrogenesis). Descriptions of the myeloarchitecture and angioarchitecture are cited only from papers by Vogt, Kawata and Pfeiffer.

Otto Marburg.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

Ophthalmological Society of the United Kingdom

President: Mr. T. Harrison Butler, 81 Edmund St., Birmingham, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.
Time: April 20-22, 1939.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Oxford Ophthalmological Congress

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Time: July 6-8, 1939.

Palestine Ophthalmological Society

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

Polish Ophthalmological Society

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

Société Française d'Ophtalmologie

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

Society of Swedish Ophthalmologists

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

Tel Aviv Ophthalmological Society

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

Tsinan Ophthalmological Society

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung, China.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

American Medical Association, Scientific Assembly, Section on Ophthalmology

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

American Academy of Ophthalmology and Otolaryngology, Section on Ophthalmology

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

Canadian Medical Association, Section on Ophthalmology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. Edward Clark, 1305-14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501-7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

Colorado Ophthalmological Society

President: Dr. Edward Jackson, 1008-A Republic Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 6:30 p. m., third Saturday of each month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

Louisiana-Mississippi Ophthalmological and Otolaryngological Society

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

Minnesota Academy of Ophthalmology and Otolaryngology

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. F. C. Smith, 106 W. 7th St., Charlotte.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Place: Statesville. Time: Sept. 21, 1939.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.
Place: Fargo. Time: May 1939.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St., N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Otolaryngology

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

Minneapolis Ophthalmological Society

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

Montgomery County Medical Society

Chairman: Dr. P. H. Kilbourne, Fidelity Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

NEW ORLEANS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Pl., Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

OPHTHALMOLOGICAL SOCIETY OF THE UNIVERSITY OF PITTSBURGH

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

PASSAIC-BERGEN OPHTHALMOLOGICAL CLUB

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

RICHMOND OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

St. Louis Ophthalmic Society

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

Toronto Academy of Medicine, Section of Ophthalmology

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

Washington, D. C., Ophthalmological Society

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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BILATERAL ENDOPHTHALMITIS COMPLICATING PNEUMOCOCCIC SEPTICEMIA

REPORT OF A CASE

JACOB REBER, M.D.

AND

HAROLD G. SCHEIE, M.D.

PHILADELPHIA

We are presenting this case of bilateral endophthalmitis because it is an instance of an ocular lesion occurring as the first manifestation of a generalized septicemia. It also affords a basis for discussion of metastatic ocular infections.

REPORT OF CASE

E. P., a 48 year old man, an upholsterer, had been in good health until Dec. 1, 1937, when he stepped on a tack while at work. This was followed by a local infection and lymphangitis of the left foot and lower part of the leg, for which he was treated by his family physician. A roentgenogram taken at the time showed no involvement of bone.

The patient appeared to be making a satisfactory recovery, all local signs and symptoms having disappeared, when, on December 15, he experienced severe frontal headache and became aware of a dimness of vision in each eye. The next day the periorbital tissues became swollen, and the patient experienced some pain in the eyes. At this time he had a slight chill and rise in temperature. He was seen in ophthalmologic consultation on December 17, when the eyes showed a typical picture of endophthalmitis, there being marked edema of the lids and chemosis of the bulbar conjunctiva. The corneas were hazy and the pupils small, irregular and fixed. Many posterior synechiae were present, and the iris structures were hemorrhagic. No view of the fundus was obtainable. Vision was reduced to perception of light.

The patient was immediately admitted to the University Hospital to the service of Dr. F. H. Adler. On his admission the temperature was 103 F. rectally. General physical examination, aside from examination of the eyes, showed the following positive findings: There were a few carious teeth. The heart was enlarged to the left. A systolic murmur and thrill were made out at the apex. The spleen was enlarged. There was slight tenderness over the medial aspect of the ball of the left foot but no inflammatory signs. The past medical history

From the Department of Ophthalmology, University of Pennsylvania Hospital.

Read before the Section on Ophthalmology of the College of Physicians, Philadelphia, Oct. 20, 1938.

included typhoid fifteen years previously and pneumonia a number of years later. The family history was irrelevant.

It was felt that we were dealing with bilateral endophthalmitis of a metastatic nature, secondary to a septic focus, and the patient was studied from this point of view. The blood count showed 20,500 white cells, with 82 per cent polymorphonuclear cells. Urinalysis showed some albumin and occasional red blood cells. The Kolmer and Kahn reactions were negative. A roentgenogram of the chest showed no evidence of pulmonary consolidation. Roentgen studies of the sinuses showed bilateral clouding of the ethmoid sinuses. The consulting otolaryngologist did not consider the upper part of the respiratory tract to be a focus of infection. Examination of the spinal fluid was essentially negative. A blood culture taken on the patient's admission was reported to be positive for the type XVI pneumococcus, thus corroborating the initial impression of septicemia.

The patient failed progressively. The temperature assumed the appearance of a septic type, with the peaks gradually rising from 101 to 105 F. The inflammatory condition about the eyes subsided gradually, the chemosis disappeared, and the eyeballs began to undergo phthisic changes, vision being reduced to perception of light.

Treatment consisted first of the administration of sulfanilamide, the patient receiving a total of 390 grains (25.27 gm.) over a period of ten days. He was given daily transfusions of 200 cc. of citrated blood. Ethylhydrocupreine hydrochloride was also administered by mouth with the hope that it might have a specific effect on the pneumococcus.

Therapy was of no avail. The blood remained consistently positive for pneumococci, many colonies being isolated on each culture. A culture taken from the conjunctiva was negative. A culture from the nose grew Staphylococcus aureus. On Jan. 2, 1938, auricular fibrillation developed, and the patient died on January 7.

At autopsy the following significant changes were observed: multiple infarctions of the spleen and kidneys; patchy bronchopneumonia of the lungs; some soft vegetations on the mitral valve, representing a recent endocarditis, and phlebitis of the left femoral vein, probably secondary to the infection in the foot. Obviously this was the septic focus.

The left eye was removed, aspiration being done previously. The fluid obtained for culture was positive for the type XVI pneumococcus. The pathologic report on the globe follows: Grossly, the eye was rather small and distorted and was filled with a white exudate. Microscopic examination showed a purulent focus in the posterior part of the choroid; a purulent exudate with numerous macrophages had separated the retina, some of which was destroyed, and almost filled the vitreous; here the choroid was not purulent, and there were lymphocytes and plasma cells in its meshes, a septic choroiditis. Other changes observed were edema of the ciliary body; numerous leukocytes pouring from the ciliary epithelium; posterior synechia; a fibrous pupillary membrane; peripheral anterior synechia; optic neuritis, and septic thrombi in the arterioles of the retina. A diagnosis of metastatic purulent endophthalmitis was made.

COMMENT

Ocular infections of metastatic origin are, of course, not infrequent, and the case reported is unusual only from the standpoint of bilateral involvement and also because the ocular signs and symptoms constituted the first warning of the existence of the infection of the blood stream.

Metastatic panophthalmitis and endophthalmitis have been reported by various authors in the literature and ascribed to various foci.

Sherer [1] stated that systemic infections may cause any one of the following manifestations in the eye: (1) iridocyclitis, (2) metastatic ophthalmitis, (3) septic retinitis of Roth and (4) ring abscess of the cornea. He cited Groenouw, who collected 166 cases of metastatic lesions in the eye due to general sepsis. Of the cases of bilateral involvement in this series, 85 per cent were fatal. Bilateral involvement was present in 27 of Leber's series of 67 cases. Friedenwald and Rones [2] studied 32 cases of septicemia, routinely removing one eye at autopsy in each case. They found focal areas of choroiditis in 9 eyes in which no signs of ocular involvement had been noted during life. The offending organism in 1 case of endocarditis and septicemia was the type II pneumococcus.

Brazeau's [3] case was one of panophthalmitis secondary to an acute empyema of the gallbladder. Laval [4] described 2 cases of metastatic panophthalmitis resulting from carbuncles of the face, Staph. aureus being isolated. Hulka [5] described a case of pneumonia complicated by metastatic uveitis. In Levine's [6] case of metastatic ophthalmitis due to Bacillus coli the primary focus was a pyonephrosis.

Of all the cases cited, that reported by Conway [7] is the most analogous to ours. His patient was a coal miner who sustained a crushing injury to a finger. This led to the development of a general infection and bilateral endophthalmitis, followed by pneumonia and death. No blood culture was reported, but it would seem that the patient undoubtedly had a pneumococcic septicemia.

The physical factor for the production of the septic choroiditis or retinitis, as the case may be, is the lodging of circulating organisms in either a ciliary or a retinal artery. These infected emboli set up a localized purulent inflammation, which, if it involves the coats of the eyeball and the surrounding orbital tissues, often results in spontaneous rupture of the sclera.

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1. Sherer, J. W.: J. Missouri M. A. **24**:294 (July) 1927.

2. Friedenwald, J. S., and Rones, B.: Some Ocular Lesions in Septicemia, Arch. Ophth. **5**:175 (Feb.) 1931.

3. Brazeau, G. N.: Am. J. Ophth. **10**:685 (Sept.) 1927.

4. Laval, J.: Metastatic Panophthalmitis from Pyogenic Cutaneous Infections, Arch. Ophth. **18**:104 (July) 1937.

5. Hulka, J. H.: Metastatic Pneumococcic Uveoscleritis Following Pneumonia, Arch. Ophth. **17**:127 (Jan.) 1937.

6. Levine, J.: Metastatic Bacillus Coli Panophthalmitis from Calculus Pyonephrosis, Arch. Ophth. **3**:410 (April) 1930.

7. Conway, J. A.: Glasgow M. J. **118**:266 (Oct.) 1932.

Of the foregoing citations, only the most recent papers contained a report of bacteriologic studies. Lundsgaard [8] reviewed the role of the pneumococcus in connection with ophthalmic conditions. He stated that, generally speaking, a pneumococcic infection of the conjunctiva is acute and benign; of the lacrimal passages, chronic and fairly benign; of the cornea, acute and malignant. This review is largely concerned with corneal ulcer.

Newman [9] made a bacteriologic study in 100 cases of ocular disease. In his series there were 3 cases of panophthalmitis, the condition in 2 being due to the type III and type X pneumococcus and in 1 to Streptococcus viridans. Newman compiled a table showing the types of pneumococcus demonstrated in ocular disease by a large series of authors. He showed that only 25 per cent of the pneumococci occurring in ocular disease belong to types I, II and III and that 75 per cent are of type IV. As is known, type IV is divided into many subtypes, of which type XVI, the organism isolated in our case, is one. In all, there are 32 culturally distinct types of pneumococcus, for many of which therapeutic serum is available.

Unfortunately, no such serum was commercially obtainable for our patient. Since it is known that ethylhydrocupreine hydrochloride is lethal for the pneumococcus in vitro, this drug was utilized in our case. In the presence of the extensive infection of the blood stream, it was without effect.

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8. Lundsgaard, K. K.: Tr. Ophth. Soc. U. Kingdom **47**:294, 1927.

9. Newman, E. W.: Diplococcus Pneumoniae and Streptococcus Viridans in Ocular Diseases: Report of One Hundred Cases, Arch. Ophth. **19**:95 (Jan.) 1938.

THE RICKETTSIA QUESTION IN TRACHOMA

I. MICROSCOPIC OBSERVATIONS ON THE VIRUS

A. E. BRALEY, M.D.
IOWA CITY

Trachoma, a protean disease of the conjunctiva and cornea, has been known since the time of Hippocrates. Hirschberg [1] in 1904 stated that entire libraries have been written concerning the disease. Scientific experiments have been carried on for over a century, but trachoma still remains a dark chapter in ophthalmology.

Opinions as to the etiologic factors, the pathologic process and clinical picture of trachoma have been so contradictory that it is impossible to believe all that is written concerning the disease. Before the discovery of the inclusion bodies by Halberstädter and Prowazek [2] many micro-organisms found in the conjunctiva were suspected of being causative agents. With this momentous discovery, new impetus was gained and many publications appeared. In 1928 Noguchi [3] described a small rodshaped micro-organism which he named Bacterium granulosis; this organism caused a follicular conjunctivitis in monkeys which was thought to be trachoma; however, it did not cause trachoma in human subjects (Wilson; [4] Proctor, Finnoff and Thygeson; [5] Thygeson; [6] Nicolle and Lumbroso,[7] and others).

In 1930 Weiss reviewed the causation of trachoma and eliminated all previously described etiologic agents, including Bact. granulosis and the Halberstädter-Prowazek inclusion body.

From the Department of Ophthalmology, College of Medicine, State University of Iowa.

This report is part of a study being conducted under a grant from the John and Mary R. Markle Foundation.

1. Hirschberg, J.: Klin. Jahrb. **13**:389, 1904.

2. Halberstädter, K., and von Prowazek, S.: Deutsche med. Wchnschr. **33**: 1285, 1907.

3. Noguchi, H.: J. Exper. Med. (supp. 2) **48**:1, 1928.

4. Wilson, R. P.: Brit. J. Ophth. **15**:433, 1931.

5. Proctor, F. I.; Finnoff, W. C., and Thygeson, P.: Am. J. Ophth. **15**:206, 1932.

6. Thygeson, P.: Role of Bacterium Granulosis in Trachoma, Arch. Ophth. **10**:1 (July) 1933.

7. Nicolle, E., and Lumbroso, V.: Arch. Inst. Pasteur de Tunis **20**:239, 1931

In 1935 Thygeson, Proctor and Richards [8] produced typical trachoma in a normal eye with material filtered through a graded collodion membrane with an average pore diameter of 0.6 micron. This result, associated with the facts that bacteria-free trachomatous material was capable of inducing infection in monkeys (Julianelle and Harrison [9]) and that bacteria plays a negative role in trachoma,[10] leads one to believe that trachoma is caused by a filtrable substance. Thygeson [11] in 1936 stated that "recent studies support the conclusion that trachoma is a virus disease" and that "evidence has been advanced which identifies the elementary body of Halberstädter and Prowazek with trachoma virus." The question of the elementary body being the trachoma virus has been the subject of a great many publications. Opinions have been divided, but at the present time it is believed that the elementary body represents the virus.

In 1933 Busacca [12] first described minute bodies in the cytoplasm of conjunctival cells from trachomatous material which he called rickettsias. Later, similar findings were reported by Cuénod and Nataf,[13] Foley and Parrot [14] and Poleff.[15] Thygeson [16] examined stained specimens of Cuénod and Nataf and both stained and unstained specimens of Busacca but was unable to demonstrate bodies that were similar to rickettsias. He concluded that the minute bodies observed were not the elementary bodies but cell granules and cytoplasmic débris. Grüter [17] considered the "rickettsias of trachoma" to be inflammatory proliferations and divisions of granules occurring normally in epithelial cells.

MATERIAL AND METHOD OF STUDY

A comparative study of smear preparations fixed in absolute methyl alcohol and stained by the method of Giemsa [18] was made of the epithelial cells from normal, nontrachomatous and trachomatous conjunctivas. Conjunctivas which did not give rise to any subjective or objective symptoms were considered normal. Conjunc-

8. Thygeson, P.; Proctor, F. I., and Richards, P.: Am. J. Ophth. **18**:811, 1935.

9. Julianelle, L. A., and Harrison, R. W.: Tr. Am. Acad. Ophth. **40**:221, 1935.

10. Stewart, F. H., in Eighth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, Schindler's Press, 1934, p. 142. Thygeson, P.: Arch. Inst. Pasteur de Tunis **22**:157, 1933.

11. Thygeson, P.: Am. J. Ophth. **19**:649, 1936.

12. Busacca, A.: Klin. Monatsbl. f. Augenh. **91**:277, 1933.

13. Cuénod, A.: Arch. d'opht. **52**:145, 1935. Cuénod, A., and Nataf, R.: ibid. **52**:573, 1935.

14. Foley, H., and Parrot, L.: Compt. rend. Soc. de biol. **124**:230, 1937.

15. Poleff, L.: Arch. d'opht. **53**:882, 1936.

16. Thygeson, P.: Problem of Rickettsias in Trachoma, Arch. Ophth. **20**:16 (July) 1938.

17. Grüter, W.: Rev. internat. du trachome **15**:9, 1938.

18. From 15 to 20 drops of Giemsa stain is added to 40 cc. of neutral distilled water, and the preparation is stained for one hour at 37 C.

tivas with acute or chronic bacterial conjunctivitis but showing no inclusion bodies were considered nontrachomatous. Conjunctivas with changes incident to exposure, e. g., ectropion, were also considered nontrachomatous. A diagnosis of trachoma was made when clinically the upper tarsus and retrotarsal fold showed follicles or papillary hypertrophy with an accompanying pannus of the cornea and when microscopic study of smears showed the Halberstädter-Prowazek inclusion bodies.

Normal conjunctival epithelium was removed, suspended in Tyrode's solution and studied immediately under bright and under dark field illumination. These cells were compared with epithelial cells obtained in a similar manner from trachomatous conjunctivas.[19]

RESULTS

Epithelium removed from normal conjunctivas and from conjunctivas with nontrachomatous inflammation when stained by Giemsa's method showed two types of granules in the cytoplasm.

The granules of one type (fig. 1 *A*) varied in size and shape and were irregularly scattered throughout the cytoplasm. In the deeper cells of the conjunctiva the granules were small and dustlike (fig. 1 *B*), while in the more superficial layers they tended to be larger and more discrete. The dustlike particles in the cytoplasm usually stained blue with the Giemsa method and in some instances gave to the cytoplasm a uniform pale blue appearance. When these granules were compared with those found under dark field illumination (fig. 1 *C*) they showed similar distribution and structure. The granules disappeared on the addition of dilute acetic acid and therefore undoubtedly were mitochondria.

The other type of granules, especially those seen in the superficial epithelial cells, were larger and did not disappear on the addition of dilute acetic acid (fig. 1 *D*). Such granules are undoubtedly closely allied to so-called keratin. With the Giemsa method these granules usually stained a pale pink (fig. 1 *E*) but were sometimes purple, the color depending on the hydrogen ion concentration of the staining solution. These larger granules of the conjunctival cells were more numerous in trachomatous material than in epithelium from the normal conjunctiva. If such granules are closely allied to keratin, they should be more numerous in the trachomatous than in the normal conjunctiva because in the former the epithelium becomes thickened and the surface cells may desquamate. These cells also showed a marked tendency to keratinize, while the normal conjunctival epithelium contained only small amounts of keratin. The tendency to extensive keratinization was also found in cases of long-standing chronic conjunctivitis and

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19. The trachomatous material was obtained at Fort Apache, Ariz., on the Fort Apache Indian Reservation, through cooperation with the Health Division, Office of Indian Affairs, Department of the Interior, Washington, D. C.

especially in cases of ectropion. Dark field examination of the epithelial cells in the latter cases showed large amounts of coarse mitochondria in the cytoplasm. These granules were similar to the bodies described as rickettsias by Busacca,[12] Cuénod and Nataf[13] and others.

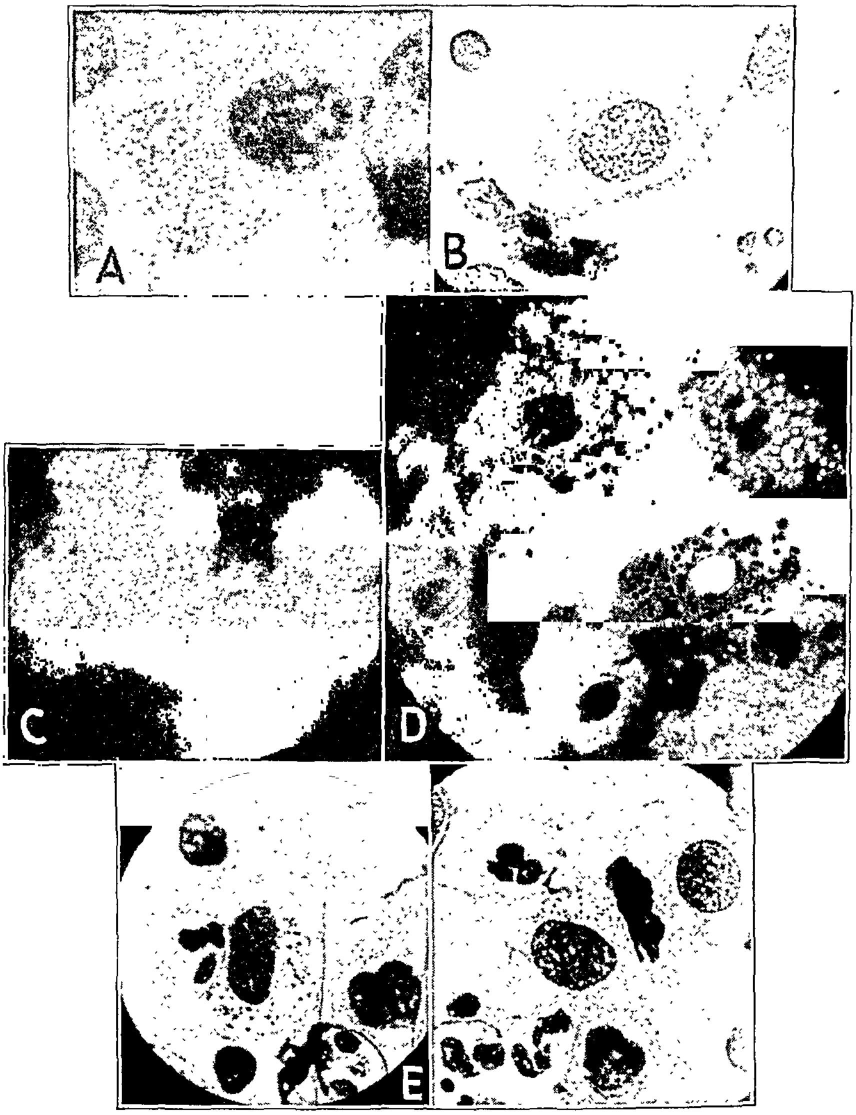

Fig. 1.—*A,* stained mitochondria from normal conjunctiva. *B,* dustlike granules in normal conjunctiva. *C,* dustlike granules in the dark field. *D,* keratin granules in the dark field. *E,* stained keratin granules.

When an elementary inclusion body was seen in a living cell in the bright field, it appeared as an indiscrete granular mass capping the nucleus (fig. 2 *A*). The mass was irregular in outline and appeared to be a part of the cytoplasm; however, by proper adjustment of the transmitted light, the outline could be distinguished. The individual

granules, while indistinct, appeared uniform. When the inclusion was seen in the dark field, it reflected so much light that it appeared homogeneous (fig. 2 *B*) and the individual granules could not be made out with certainty. Such masses differ materially from masses of mitochondria in that the elementary inclusion body is globular while the mitochondria assumes weird, constantly changing, fantastic shapes. According to Rötth,[20] the elementary body is also insoluble in dilute acetic acid.

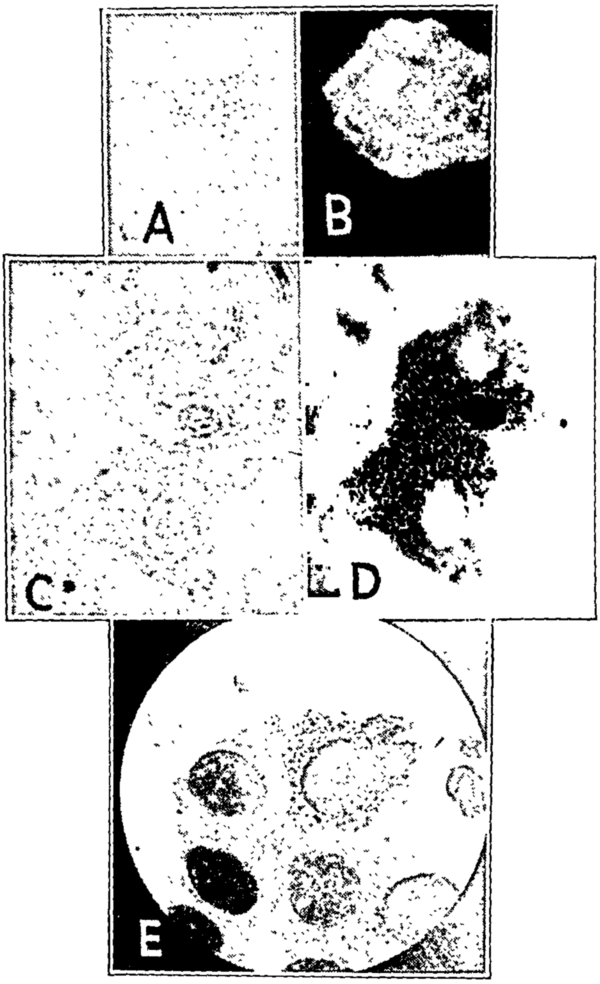

Fig. 2.—*A,* elementary inclusion body in the bright field. *B,* elementary inclusion body in the dark field. *C,* initial inclusion bodies in the bright field. *D,* initial inclusion bodies in the dark field. *E,* scattered elementary bodies.

The initial inclusion body, as seen in the bright field, appeared as a small discrete granular mass (fig. 2 *C*). Such bodies may lie in any position in the cytoplasm. In the dark field the inclusion was composed of brilliantly lighted spherical or oval granules (fig. 2 *D*). Mitochondria or keratin granules in the cytoplasm may be confused with the initial inclusion body when studied in the dark field; however, in my study

20. Rötth, A.: Arch. f. Ophth. **128**:381, 1932.

mitochondria and keratin granules were not seen when the cell was illuminated by direct light, and the initial inclusion bodies were distinct.

The virus of psittacosis is identified easily in dark field preparations.[21] The size of the virus is approximately the same as the measured size of the elementary body of trachoma. In the dark field, the inclusion of psittacosis appears similar to the inclusion of trachoma.

Rice [22] and, later, Thygeson [23] demonstrated that the matrix of the inclusion stains with iodine. Dilute or undilute compound solution of iodine stains the inclusion a deep reddish brown, while the remainder of the cell and other cells stain a pale yellow. In my preparations normal epithelial cells (fig. 1 *C*) when stained with dilute compound solution of iodine became uniformly pale yellow. The mitochondria were not visible in the bright field after staining with iodine but could be seen in the dark field provided the iodine was sufficiently dilute. In the dark field the elementary inclusion body did not reflect the light after staining with iodine, undoubtedly because the stained matrix absorbs light.

At times true elementary inclusion bodies of trachoma were found scattered throughout the cell (fig. 2 *E*). One could confuse these with mitochondria or keratin granules, but on close observation several essential differences were noted. In a cell filled with true elementary bodies the matrix was clear and unstained, while mitochondria and keratin granules lay in a pale blue or pink matrix. Also true elementary bodies were uniform in size and shape and stained uniformly, while the mitochondria and keratin granules varied in size, shape and staining reactions (figs. 1 *A* and 2 *E*).

Whether the etiologic factor of trachoma should be classified as a filtrable virus or as a rickettsia cannot be definitely settled by study of microscopic preparations, but from observations on the trachoma virus it seems to be more closely allied to the former than to the latter.

CONCLUSIONS

1. Mitochondria and keratin granules of normal living conjunctival epithelial cells as seen in the dark field can be stained by the Giemsa method.

2. The inclusion bodies of trachoma and of psittacosis are similar under dark field illumination.

3. The bodies described and photographed by Busacca, Cuénod and Nataf and others undoubtedly represent stained mitochondria and keratin granules rather than rickettsias.

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21. Bland, J. O. W., and Canti, R. G.: J. Path. & Bact. **40**:231, 1935.
22. Rice, C. E.: Am. J. Ophth. **19**:1, 1936.
23. Thygeson, P.: Am. J. Path. **14**:455, 1938.

NAEVUS FLAMMEUS ASSOCIATED WITH GLAUCOMA

REPORT OF A CASE

MORRIS H. PINCUS, M.D.

BROOKLYN

The clinical syndrome of naevus flammeus associated with glaucoma has been recognized for many years. It is a relatively rare condition. Schirmer [1] in 1860 was the first to describe this clinical entity, and since then about 75 cases have been reported. O'Brien and Porter [2] in a complete survey of the literature showed that in the majority of the cases the glaucoma was of the infantile type and generally unilateral, the affected eye in many instances being partially or completely blind.

Salus [3] was the first author to show that the glaucoma associated with naevus flammeus need not be congenital but may appear later in life. He cited 2 cases; 1 was that of a woman aged 45 and the other that of a man aged 57.

Usually, the nevus and the glaucoma occur on the corresponding side of the face, but there are exceptions to this rule. Löwenstein [4] reported a case in which naevus flammeus occurred on the left cheek of a woman of 40, who also had a partial glaucomatous excavation of the right disk but with normal tension. The visual acuity and visual field of each eye were normal.

The glaucomatous condition does not always produce partial or complete blindness. In Vögele's case [5] the nevus was on the right side of the face. The disk of the corresponding eye was deeply excavated. The intraocular pressure was increased to 47 mm. of mercury, yet the visual acuity and visual fields were normal.

Read before the Brooklyn Ophthalmological Society, Feb. 17, 1938.

From the Ophthalmologic Service of Dr. W. Moehle, Kings County Hospital.

1. Schirmer, R.: Ein Fall von Telangiektasie, Arch. f. Ophth. **7**:119, 1860.

2. O'Brien, C. S., and Porter, W. C.: Glaucoma and Naevus Flammeus, Arch. Ophth. **9**:715 (May) 1933.

3. Salus, R.: Glaukom und Feuermal, Klin. Monatsbl. f. Augenh. **71**:305, 1923.

4. Löwenstein, A., in discussion on Salus, R.: Glaukom und Feuermal, Klin. Monatsbl. f. Augenh. **70**:540, 1923.

5. Vögele, N.: Glaukom und Naevus flammeus, Klin. Monatsbl. f. Augenh. **81**:393, 1928.

Naevus flammeus and glaucoma may be associated with other bizarre clinical signs. Tyson[6] found right homonymous hemianopia, which was apparently due to a calcified plaque; it was shown roentgenographically to be situated in the region of the left occipital lobe. The tension of the left eye was 45 mm. of mercury. The cupping of the disk was increased, and the blood vessels were pushed to the nasal side. The visual acuity of each eye was 20/20.

That intracranial changes do occur in instances of naevus flammeus has been well established. A history of epileptiform seizures on the side opposite the facial nevus was obtained in 7 of the reported cases. In 5 cases calcified meningeal vessels were demonstrated roentgenographically. Vascular changes were observed in the meninges in three cases in which autopsy was performed. Hemiplegia was noted in 8 of the reported cases on the side opposite the facial nevus.

REPORT OF CASE

D. C., a white man aged 45, came to the outpatient department of the ophthalmologic service on April 20, 1937, complaining of occipital headaches. These headaches began rather insidiously a few months before examination and became progressively worse. They lasted for a number of hours and occasionally for two or three days. Concomitantly with the appearance of these headaches, the patient found it somewhat difficult to read at night. There was no history of halos or of intraocular pain.

His health prior to 1915 had been excellent. While serving with the Russian army, he contracted typhus and pneumonia. In 1919 he had influenza. For the past twelve years he had had pains and aches in both shoulder joints and in the lumbar region. There was no history of a venereal infection. The family history was noncontributory.

The patient was well developed. His heart and lungs were normal. The blood pressure was 162 systolic and 90 diastolic. Otolaryngoscopic examinations revealed nothing more than hypertrophied tonsils.

The blood chemistry and urine were normal. The Wassermann reaction of the blood was negative.

A roentgenogram of the shoulders was normal. A roentgenogram of the lower part of the spine revealed slight hypertrophic changes along the margins of the lower lumbar portion. There was a partial spina bifida occulta of the sacrum. A roentgenogram of the skull revealed the sella turcica to be somewhat shallower than normal. There was no evidence of any calcification of the intracranial vessels.

On the right side of the face, starting at the hair line and extending to the outermost portions of the upper and lower lids of the right eye, was a naevus flammeus. The lids otherwise were normal.

The corneas were clear and measured 12 mm. horizontally. The anterior chambers were normal in depth. The pupils were 3 mm. in diameter and round and regular. They reacted to direct light, consensually and in accommodation. The media were clear. Ophthalmoscopic examination of the right fundus revealed a normal-

6. Tyson, H. H.: Naevus Flammeus of the Face and Globe, Arch. Ophth. **8**:365 (Sept.) 1932.

sized disk, with its margins clearly outlined. The physiologic cup was deep, and the blood vessels were pushed over to the nasal edge. The arteries were somewhat narrowed, and the veins were somewhat fuller than normal. There was no increased tortuousity of the vessels. The macular area, as well as the remainder of the fundus, was normal. The ophthalmoscopic findings in the left fundus were similar to those in the right, except that the physiologic cup was normal in depth and a cilioretinal vessel was present.

Slit lamp examination of the right eye showed that the cornea, anterior chamber and lens were normal. The density of the stroma of the iris appeared to be increased. No blood vessels were seen. The vessels of the bulbar conjunctiva were normal, but the episcleral blood vessels were full, markedly tortuous and convoluted. These changes were particularly marked on the temporal side of the globe.

Slit lamp examination of the left eye gave similar results, but the episcleral blood vessels were not nearly as tortuous and convoluted as those of the right eye.

The visual acuity of each eye was 20/20.

At that time the intraocular tension (Schiötz) was 30 mm. of mercury in each eye. A qualitative perimetric examination revealed a slight concentrically contracted visual form and color fields in each eye, somewhat more marked in the upper and nasal portions of the right eye.

A 1 per cent solution of pilocarpine hydrochloride was prescribed, but the patient did not return for observation. Five months later he returned, again complaining of severe headaches. However, he stated: "When I used the drops, the headaches were not so severe." The visual acuity remained at 20/20. The qualitative perimetric fields were substantially the same as at the previous examination. The intraocular tension of the right eye was 35 mm. of mercury, and that of the left eye, 28 mm. of mercury. The solution of pilocarpine hydrochloride was prescribed again, and the patient was told to return when he had used all of it. When he returned a quantitative perimetric examination revealed a slight concentric contraction of the field, more marked in the upper and nasal quadrants of the right eye. The size of the blindspot of the left eye was normal. The blindspot of the right eye was enlarged laterally and superiorly, forming a beginning Seidel scotoma.

After a lapse of two months, during which time the patient was not using the solution of pilocarpine, he returned, complaining of severe headaches. The intraocular tension of the right eye was now 35 mm. of mercury and that of the left eye, 25 mm. of mercury. Fifty cubic centimeters of a 50 per cent solution of dextrose was administered intravenously, and the intraocular tension was recorded every ten minutes for one and one-half hours. The intraocular tension of the right eye varied between 32 and 38 mm. of mercury; that of the left eye varied between 22 and 28 mm. of mercury.

COMMENT

Naevus flammeus associated with glaucoma occurs in the newborn or the very young and also in much older persons. In any case, partial or complete blindness or some constrictions of the visual fields may occur. The glaucoma is chronic and noninflammatory: Oddly enough, it has been found that various pathologic intracranial changes occur in this condition.

The common clinical ocular signs observed in these glaucomatous eyes may be summarized as follows:

In the conjunctiva or episclera dilated vessels or nevi may be found. In the iris the changes may be heterochromia, increased density of the stroma or dilated blood vessels. In the choroid angiomas or nevi may be seen. In the retina there may be varicosities or tortuous blood vessels, and in the optic disk, increased cupping, alone or combined with partial or complete atrophy.

In the 15 eyes that were examined pathologically it was found that 11 showed angioma of the choroid, 1 showed angiomatous changes in the iris near the filtration angle and 1 showed an irregularity of Schlemm's canal and the scleral spur. In 2 others no changes were observed in the anterior segment or in the choroid. In all the eyes, however, the intraocular capillaries were dilated.

The ocular changes in my patient agree with those just mentioned. His eye showed tortuous and convoluted episcleral vessels, increased density of the stroma of the iris, deep cupping of the optic disk and increased intraocular tension.

This patient, therefore, belongs to the group in whom the glaucoma occurs later in life. He is not totally or even partially blind. His glaucoma is not progressing.

Chronic simple glaucoma can, of course, occur in a patient who happens to have a naevus flammeus. While it is true that it may exist for a little while without inducing subjective symptoms, this time is not of long duration. My patient has been followed for a long period without any apparent aggravation of his symptoms.

The causation of naevus flammeus is unknown. The disease is supposed to be of congenital origin. The cause for the glaucomatous state is also nebulous. There are as many theories for this condition as there are observers. I shall mention but few of them.

Elschnig [7] accounted for the glaucomatous state on the basis of a plethoric condition of the choroidal vessels. This theory, according to him, was supported by the fact that compression of the carotid artery lowered the intraocular pressure.

Yamanaka [8] was of the opinion that there were two causes: (1) a dilatation of the choroidal vessels and an increase in their number, and (2) malformation of the meshwork of the iridocorneal angle with an insufficient outflow of aqueous.

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7. Elschnig, A.: Naevus vasculosus mit gleichseitigem Hydrophthalmus, Ztschr. f. Augenh. **39**:189, 1918.

8. Yamanaka, T.: Naevus Flammeus mit gleichseitigem Glaukom, Klin. Monatsbl. f. Augenh. **78**:372, 1927.

Tyson[6] suggested that the anterior chamber is blocked by a "plasmoid" aqueous resulting from changes in capillary permeability. He used the fluorescein test in his case, and a greenish color of the aqueous of the involved eye was soon apparent, while the aqueous in the normal eye remained clear.

Dunphy[9] and Mehney[10] agreed with Tyson's theory. Dunphy stated that there is no proof that the aqueous in these cases is truly plasmoid, i. e., that its protein content approaches that of the blood. No one, so far as he knew, made any determinations on the aqueous in such cases. Nevertheless, he stated that it is known that any condition which leads to an increased protein content tends to produce an increased formation of aqueous by reducing the difference in the osmotic pressure of the blood and the aqueous. The amount of aqueous formed in any unit of time is therefore increased. He found that the injection of 50 cc. of a 50 per cent solution of dextrose intravenously reduces the intraocular tension of normal eyes by increasing the osmotic pressure of the blood but that it has no effect on the intraocular tension of glaucomatous eyes. According to him, this fact lends support to the theory of abnormal permeability.

Mehney made a chemical analysis of the aqueous in his case and found 0.032 per cent of protein as compared to the normal of 0.02 per cent. The protein content of the blood serum in his case was normal.

CONCLUSION

A case of unilateral naevus flammeus associated with glaucoma of the corresponding eye is presented. This case illustrates (1) that glaucoma need not be of the infantile type but may appear later in life; (2) that blindness, total or partial, need not be associated with this condition, and (3) that Tyson's contention, i. e., that there is a change in the capillary permeability in this disease, comes closest to a true explanation of the observed phenomenon.

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9. Dunphy, E. B.: Glaucoma Accompanying Naevus Flammeus, Am. J. Ophth. **18**:709 (Aug.) 1935.

10. Mehney, G. H.: Naevus Flammeus Associated with Glaucoma, Arch. Ophth. **17**:1018 (June) 1937.

ANGIOID STREAKS

A. HAGEDOORN, M.D.
AMSTERDAM, NETHERLANDS

REPORT OF A CASE OF THE SYNDROME OF GRÖNBLAD AND STRANDBERG [1] (ANGIOID STREAKS AND PSEUDOXANTHOMA ELASTICUM)

History.—A woman aged 48 was admitted to the neurologic ward of the Wilhelmina Hospital on March 26, 1935. During the night of March 23 she noticed a loss of power in her left arm and leg, which had developed insidiously. The next morning there was a disturbance of speech and swallowing. The patient spoke indistinctly and was able to form a few letters only with difficulty. In addition, she noticed that she had difficulty in moving her tongue while speaking. She understood all that was said to her. She complained of a tingling sensation in the affected limbs, more marked in the left leg. "Menstruation" started on March 24 and ended on March 26; the previous week, when the period was due, the patient had also menstruated. This was the first time that this function had been irregular.

Inquiry revealed that for a short time in 1932 the patient's left arm and leg had been weak, at which time there had been a disturbance in speech and swallowing. Neither fever nor chills had occurred while she was at home. During the past five years she noticed that she tired easily and was breathless, especially after climbing stairs, but that she had no difficulty from palpitation. She had no knowledge of any preceding renal disease or of ever having had acute articular rheumatism, St. Vitus' dance or a venereal disease. Questions concerning the internal organs revealed nothing unusual.

The patient came from a healthy family and until her fifth year had never been ill. She had no knowledge of ever having a cutaneous disease. She was married, and her husband was healthy. She had given birth to eight children. At the second confinement twins were born; they were weak and died shortly after birth. The fourth and fifth pregnancies ended in abortions. The patient's father died at the age of 63 (bloody stools and calcified veins). Her mother died at 63 of "old age." The patient knew of no cutaneous disease or ocular trouble existing in the family.

Physical Examination.—The patient looked ill but was compos mentis. She was dyspneic, her face was congested, and the extremities were slightly cyanotic. The pulse rate was 70 per minute, and the pulsations were regular, even, well filled and tense and equal on the two sides. The blood pressure was 210 systolic and 100 diastolic. Respiratory movements were frequent, control of which, though possible, resulted in a limited excursion. The heart was enlarged to the left, the

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Read before the Dutch Ophthalmological Society, Amsterdam, Netherlands, Dec. 13, 1936.

1. From the ophthalmologic (W. P. C. Zeeman), neurologic (B. Brouwer) and pathologic (H. T. Deelman) laboratories of the University of Amsterdam. This case was studied by Drs. Hagedoorn, J. J. G. Prick and E. Sekir, and the report was prepared by Dr. Prick.

apical beat heaving. Auscultation revealed a soft systolic souffle over all the valves. The second aortic sound was louder and more ringing than the second pulmonic sound. On the patient's admission the lungs were free of disease. Examination of the abdomen also gave negative results. The skin of the neck, axillas, chest and abdomen, as well as the flexor surface of the elbows and the folds in the groins, was typical for pseudoxanthoma elasticum. The skin was thickened, lay in folds, felt inelastic and presented violet-tinted, yellowish macular and papular efflorescences. The distribution of the cutaneous condition was symmetric. Examination of the visible mucous membranes revealed nothing abnormal. The fundus of the eye revealed a few angioid streaks and a disk-shaped degeneration of the retina. The papilla nervi optici was surrounded by a gray halo, more marked on the temporal side. Heaps of pigment were seen scattered over the fundus. Neurologic examination revealed left hemiplegia due to involvement of the central nervous system. Active movement of the left arm and leg was practically impossible, whereas the left facial muscles, especially those supplied by the lower branch of the facial nerve, were paretic. The right side of the body also showed signs of cerebral hemiparesis, though less marked, and the muscles supplied by the lingual branch of the right facial nerve were also involved. Surprisingly better was the voluntary innervation of the region supplied by the upper branches of this nerve. The bilateral musculature of the palate and pharynx was under poor control. Both palate arches moved insufficiently with intonation, the left less than the right. Owing to difficulty in swallowing, the patient choked repeatedly. Voluntary movements of the tongue were possible but limited. The tongue deviated from the midline when extended. Turning of the head and lifting of the shoulders occurred in the normal way. On the patient's admission innervation of the muscles of mastication was intact, and no trismus could be established. Ocular movements were normal, and no conjugate deviation was present. Slight nystagmus developed when the patient looked to the right and to the left. Investigation of the special senses brought out the following facts: Sharpness of vision was reduced on each side to ½ and could not be corrected. The visual fields were normal. Hearing, taste and smell were intact. In addition to the disturbance in swallowing, there existed a speech disorder, a dysarthria. Owing to poor articulation, the patient's speech was difficult to understand and in addition had an open nose quality. Spontaneous respiratory movements occurred in a normal way and without any obvious difference between the two sides of the thorax. Sensibility in the region supplied by the trigeminus nerve showed no disorder. Reaction to the vital stimuli was less sensitive in the left arm and leg as well as in the left buttock. Deep sensation was maintained. Coordination of the left arm and leg could not be judged; owing to the marked disturbance of movements, slight ataxia was present in the right arm and leg.

As has been reported, the respiratory movements could be controlled, but only to a limited degree. Therefore no obvious difference between the left and the right side of the thorax could be established. The reflexes on admission were as follows: The pupils reacted to light and in convergence. The corneal and masseteric reflexes were positive. A positive facial reflex was present on the left side. The pharyngeal reflex was, however, absent. No pathologic bulbar reflexes could be elicited. The biceps and triceps reflexes were increased on each side, the left more than the right. The reflexes of Mayer and Leri as well as the abdominal reflex were absent on both sides. The reflexes of Babinski and Chaddock were positive on both sides. A slight hypertonia of the musculature of the arms

and legs existed, more marked on the left. No psychic disturbance could be found. Compulsion laughing and compulsion crying were both absent.

The day after the patient's admission flaccid paralysis of the muscles of mastication developed, and the masseteric reflex had disappeared. During the night of March 28 a curious complication developed in the form of acute bleeding from the gastrointestinal tract. The patient suddenly vomited blood, became pallid and sweated profusely; the pulse beat was small and frequent. Examination of the abdomen revealed no "defense." The gastric region was distended but not tender. Nothing could be found that might lead to a diagnosis of ulcer, carcinoma or hepatic cirrhosis. The following day signs of lobar pneumonia developed. The patient died on April 1.

Laboratory Tests.—The urine contained albumin, hyaline casts and a few clusters of leukocytes. The specific gravity was 1.022. The urea content of the blood serum was 300 mg. per liter (Ambard). The Wassermann and the Sachs-Georgi reaction of the blood were negative. Lumbar puncture was not performed.

The vomitus in addition to blood contained free hydrochloric acid. The feces gave a strongly positive benzidine reaction. The sedimentation rate was 9 mm. in one hour. The blood picture was as follows: hemoglobin (Sahli), 60 per cent (corrected value); leukocytes, 2,200; erythrocytes, 3,900,000; eosinophils, 0; basophils, 0; polymorphonuclears, 4 per cent; segmented polymorphonuclears, 89.5 per cent; lymphocytes, 3.5 per cent, and large monocytes, 3 per cent. On March 29 the hemoglobin content (Sahli) was 48 per cent (corrected value).

Diagnosis.—A clinical diagnosis was made of pseudobulbar paralysis; pseudoxanthoma elasticum, with an associated disease of the fundus; essential hypertension; cardiac hypertrophy and dilatation; hematemesis (cause?), and lobar pneumonia.

GENERAL INTRODUCTION

Grönblad's [1a] statement that pseudoxanthoma elasticum is often associated with angioid streaks (Herman Knapp, 1892) has been repeatedly confirmed. Shortly after the publication of Grönblad, Marchesani and Wirz [2] described the simultaneous occurrence of pseudoxanthoma and angioid streaks.

It should be stated that the simultaneous occurrence of pseudoxanthoma with a disease of the fundus oculi had already been called attention to in a case reported by Hallopeau and Laffitte [3] in 1903. Antonelli, the ophthalmologist who examined the patient, failed, however, to recognize the relation between central chorioretinitis and secondary atrophy of the disk with angioid streaks, which, as figure 1 shows, may have been practically impossible at that time. Hallopeau and Laffitte stated: "Y a-t-il une relation entre ces altérations oculaires

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1a. Grönblad, E.: Angioid Streaks—Pseudoxanthoma Elasticum: Vorläufige Mitteilung, Acta ophth. **7**:329, 1929; Angioid Streaks—Pseudoxanthoma Elasticum: Der Zusammenhang zwischen diesen gleichzeitig auftretenden Augen- und Hautveränderungen, ibid. (supp. 1) **10**:1, 1932.

2. Marchesani, O., and Wirz, F.: Die Pigmentstreifenerkrankung der Netzhaut, Arch. f. Augenh. **104**:522, 1931.

3. Hallopeau and Laffitte: Nouvelle note sur un cas de pseudoxanthome élastique, Ann. de dermat. et syph. **4**:595, 1903.

et le pseudoxanthome? En l'absence d'antécédents et de signes de syphilis ainsi que d'albuminurie et de glycosurie, cette hypothèse est la plus vraisemblable." (Is there a relation between these ocular changes and pseudoxanthoma? In the absence of a history or symptoms of syphilis, such as albuminuria and glycosuria, this hypothesis seems reasonable.) Thus they were the first to detect the relation between pseudoxanthoma and ocular disease.

The fundi of the eyes of this patient could not be studied thoroughly because of her clinical condition; she died a few hours after the ocular examination. It was not possible to make an accurate drawing or a photograph of the fundi. There was marked degeneration of the circumpapillary and the macular region, but it was only just possible to trace a few streaks or lines, which resembled angioid streaks.

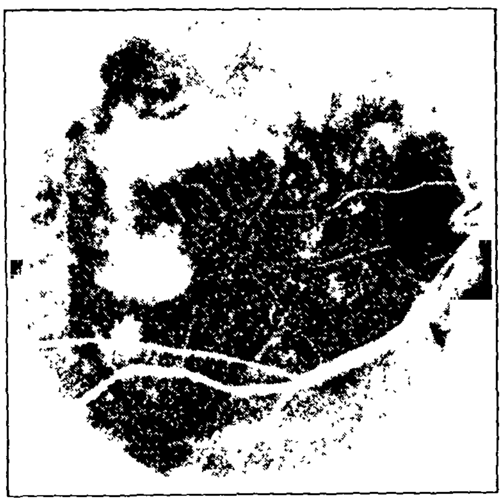

Fig. 1.—Angioid streaks. There is extensive degeneration in the macular and circumpapillary region. The angioid streaks are difficult to trace.

These macular lesions, which usually appear in the later stages of the disease, have often been compared with senile disciform degeneration of the macula (Bonnet [4]).

Figure 1 is a picture of the fundus in a similar case in which the condition was less advanced. The patient had six children who showed no signs of angioid streaks. An eye of one child contained a coloboma of the iris and choroid.

During the last few years several articles dealing with angioid streaks have appeared in the American literature (Dykman,[5] Goedbloed [6] and Benedict [7]). Therefore, the ophthalmoscopic picture does not need introducing. Certain details will be considered later. It will be remembered

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4. Bonnet, P.: Evolution des altérations de la macula dans les "stries angioides de la rétine," Arch. d'opht. **52**:225-240, 1935.

(Footnotes continued on next page)

that the streaks may be red; they may also be gray or of varying shades of brown to almost black. The edges more often have the appearance of torn paper than the smooth straight or curved boundaries of a wall of a vessel. Figures 2, 3, 6 and 7 demonstrate the variability of the fundus picture of angioid streaks.

In the cases mentioned in this paper the clinical and histologic diagnosis of pseudoxanthoma was made or confirmed by Dr. W. L. L. Carol, professor of dermatology at the University of Amsterdam.

HISTOLOGIC PICTURE OF ANGIOID STREAKS AS DESCRIBED IN THE LITERATURE

The eye described by Lister [8] was seriously damaged and cannot be accepted as one affected with angioid streaks. Magitot [9] gave a histologic

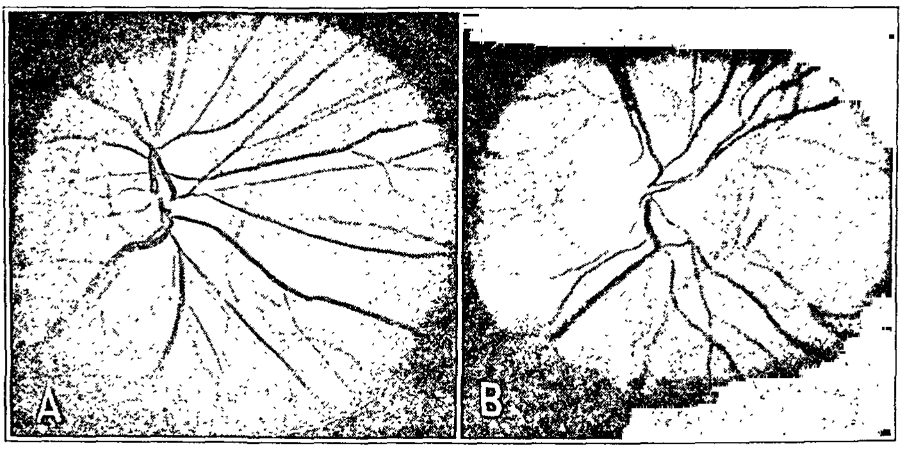

Fig. 2.—Angioid streaks. In *A* the streaks are readily seen. There is no secondary macular degeneration. Note the gray shade of the fundus and the absence of choroidal vessels. Confusion of the streaks with choroidal vessels is impossible. In *B* the streaks are readily seen, but confusion with choroidal vessels is possible.

description of Siegrist's [10] pigmented streaks in arteriosclerosis of the choroid. The changes observed by Verhoeff [11] in an eye damaged by

5. Dykman, A. B.: Angioid Streaks of the Retina: A Report Concerning Two Cases Associated with Pseudoxanthoma Elasticum, Arch. Ophth. **11**:283 (Feb.) 1934.

6. Goedbloed, J.: Syndrome of Groenblad and Strandberg: Angioid Streaks in Fundus Oculi, Associated with Pseudoxanthoma Elasticum, Arch. Ophth. **19**: 1 (Jan.) 1938.

7. Benedict, W. L.: The Pathology of Angioid Streaks in the Fundus Oculi, J. A. M. A. **109**:473 (Aug. 14) 1937.

8. Lister, W. T.: Angioid Streaks of the Retina, Ophth. Rev. **22**:151, 1903.

9. Magitot, A.: Pigmentation angioide de la rétine, Ann. d'ocul. **145**:2, 1911.

(Footnotes continued on next page)

iritis, glaucoma and iridectomy, with loss of vitreous, cannot, as Franceschetti and Roulet [12] concluded, be accepted as typical for the anatomic substrate of angioid streaks; ophthalmoscopic examination was impossible in this case. In the other eye of Verhoeff's patient a gray zone surrounding the optic disk was seen, from which there extended two white streaks. Over a portion of the fundus of the enucleated eye, dark "angioid streaks" were seen macroscopically, which on microscopic examination were revealed as being parts of the inner layers of the choroid, thrown into ridges by a cicatricial contraction of fibrous tissue, the latter having replaced the deeper layers. Such a histologic picture does not fit in with the clinical findings; moreover, the diagnosis of true angioid streaks in the other eye cannot be considered as definitely proved. Wilmer [13] reported a case in which investigation was incomplete. Benedict [7] in a recent paper reported that he was unable to find any specific histologic lesion. Probably this was due to the way in which the sections were made. Law [13a] identified angioid streaks with the retinal folds he found in his interesting case (with gastric hemorrhage).

It may be concluded that a histologic description of angioid streaks is still lacking, though on the basis of ophthalmoscopic studies it seems probable that the primary site of the condition must be found in Bruch's membrane (Grönblad). Böck's [13b] observations are in accordance with my findings in 1936.

DIFFERENTIAL DIAGNOSIS

Angioid streaks, no matter how varied in shape and color they may be, are generally so typical that but few conditions can give rise to difficulties in differential diagnosis. Confusion with choroidal vessels (fig. 4) is not likely to occur if the aspect of angioid streaks is familiar to the observer. Siegrist's pigmented streaks in arteriosclerosis of the choroid may simulate angioid streaks, but their rough, irregular, darkly

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10. Siegrist, A.: Beitrag zur Kenntnis der Arteriosklerose der Augengefässe, Internat. Ophth. Kong., Utrecht, 1899, p. 131.

11. Verhoeff, F. H.: The Nature and Pathogenesis of Angioid Streaks in the Ocular Fundus, Tr. Sect. Ophth., A. M. A., 1928, p. 243.

12. Franceschetti, A., and Roulet, E. L.: Le syndrome de Groenblad et Strandberg (stries angioïdes de la rétine et pseudoxanthome élastique) et ses rapports avec les affections du mésenchyme, Arch. d'opht. **53**:401, 1936.

13. Wilmer, W. H.: Angioid Streaks of the Choroid, in Atlas Fundus Oculi, New York, The Macmillan Company, 1934, fig. 92; cited by Franceschetti and Roulet.[12]

13a. Law, F. W.: A Contribution to the Pathology of Angioid Streaks, Tr. Ophth. Soc. U. Kingdom **58**:191, 1938.

13b. Böck, J.: Zur Klinik und Anatomie der gefässähnlichen Streifen im Augenhintergrund, Ztschr. f. Augenh. **95**:1, 1938. His paper appeared after this report had been submitted for publication, so that it cannot be discussed as it deserves.

pigmented aspect is seldom observed in angioid streaks and never over the entire course of the streaks. The condition in Ejler Holm's [14] cases, described as angioid streaks, was of this type.

Striate retinitis must also be borne in mind. Under this heading various conditions have been described, even angioid streaks. Striate retinitis is described as following detachment of the retina. The streaks are not so sharply marked off, are not red and lack the peculiar torn-paper aspect of angioid streaks; there are generally other rests of detachment present (pigmentation) or at least a history of detachment. The term striate retinitis is found in "Kurzes Handbuch der Ophthalmologie" but not in Parson's textbook, "Pathology of the Eye." Rönne [15] reported finding a strand of connective tissue between the retina and the choroid in a case of striate retinitis; other authors have expressed the belief

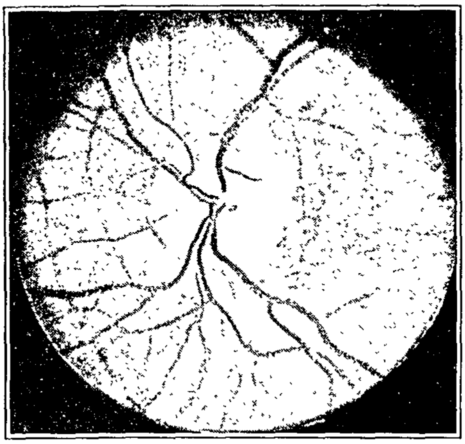

Figure 3

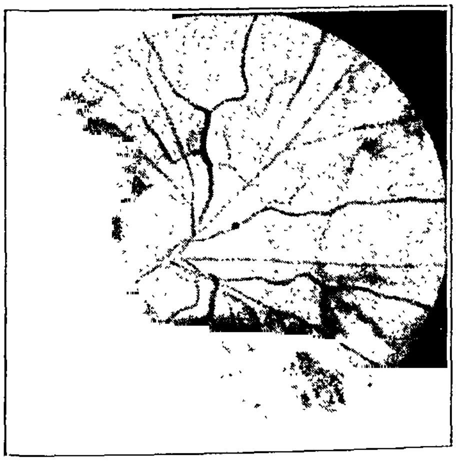

Figure 4

Fig. 3.—Angioid streaks. Smaller streaks resembling a lake district are seen bordering the disk.

Fig. 4.—Myopic fundus. The choroidal vessels simulate angioid streaks.

that the strands are ridges in the choroid. Striae, typical of detachment, have been described by Leber.[16] Figure 5 probably represents the anatomic substrate of such a stria, which is entirely different from the pigmented streaks, marking off an old detachment. It was found in one of the eyes described by Van der Meer.[17] In addition,

14. Holm, E.: On the Site of the Angioid Streaks in the Fundus Oculi, Acta ophth. **2**:152, 1924; Pigmented Chorioidal Vessels, ibid. **5**:188, 1927.

15. Rönne, H.: Zur Anatomie der Streifenbildung nach Netzhautablösung, Arch. f. Ophth. **75**:284, 1910.

16. Leber, in von Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, p. 1442, fig. 230.

17. Van der Meer, B. C. J.: Over de operatieve behandeling van netvliesloslating, Maastricht, Netherlands, Stols, 1935.

other varieties of streaklike pigmentations and demarcations may occur in cases of older retinal detachment.

The whitish "atrophic" appearance of the circumpapillary region may dominate the picture in cases of angioid streaks (fig. 6), the streaks being

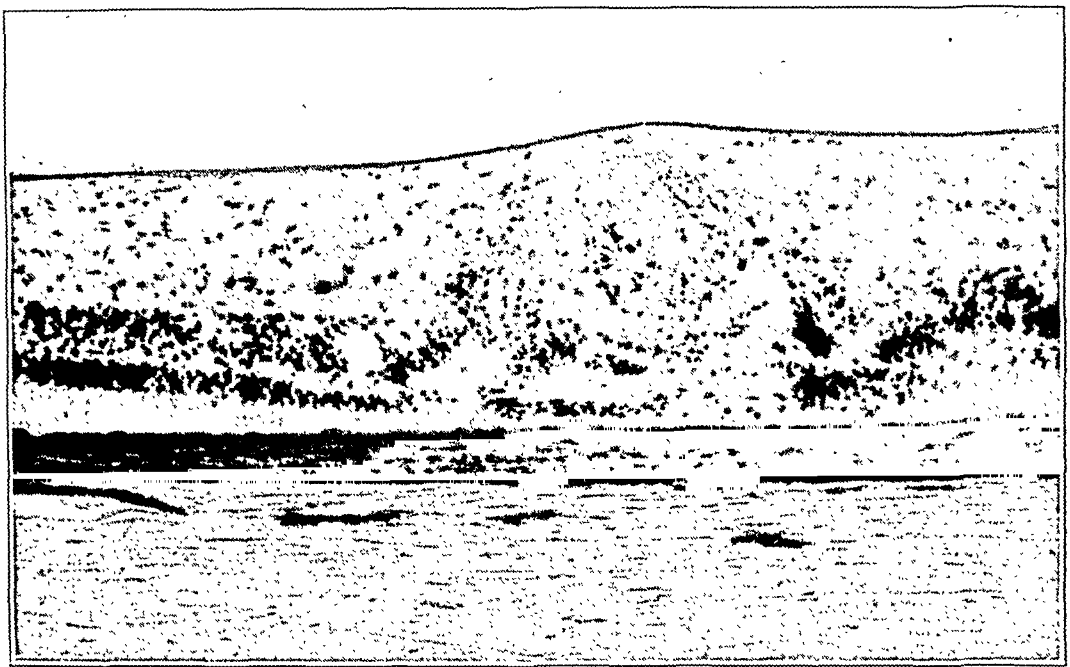

Fig. 5.—Retinitis striata; cross section through the retinal stria in a case of retinal detachment. Van Gieson's stain; × 122.

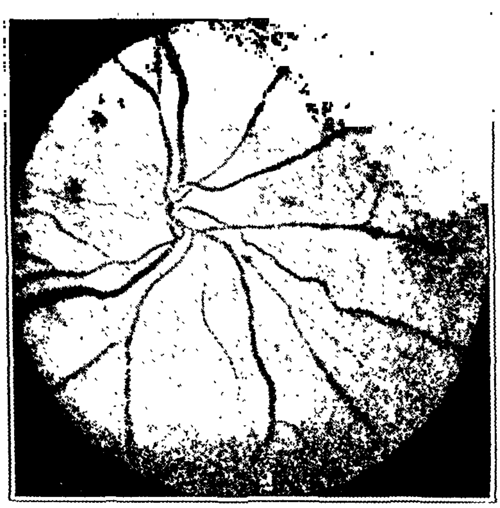

Fig. 6.—Angioid streaks, with involvement of the circumpapillary region. The streaks are less generalized but are readily seen.

visible only on careful examination. (They were even difficult to trace in the fundi showing figure 7. In a similar case but one in which the condition was less advanced the diagnosis remained doubtful, since the cutaneous condition was absent and definite angioid streaks could not be

traced [fig. 8]). In myopia the circumpapillary region may also be white and irregularly pigmented, but the sharp line of demarcation of the atrophic areas at the posterior pole is different from the gradual fading of the prominent areas of degeneration ("pseudotumor"), as is seen in angioid streaks and in advanced stages of Junius and Kuhnt's

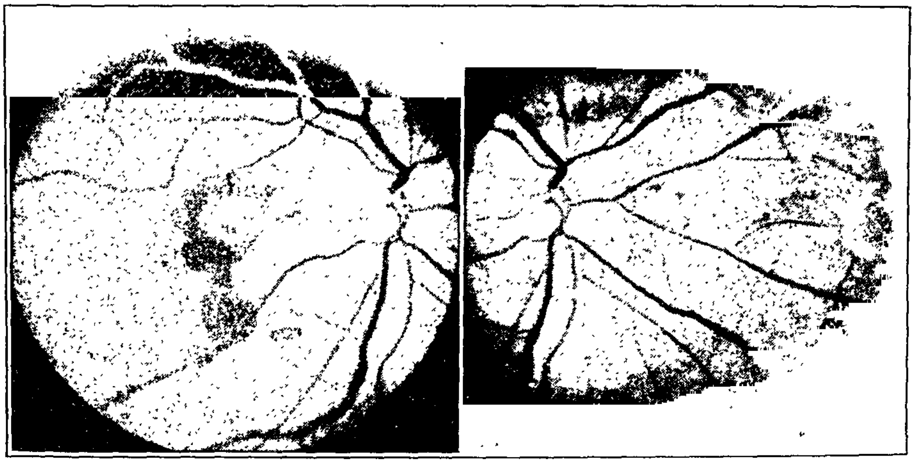

Fig. 7.—Angioid streaks, in a case of pseudoxanthoma of the skin with grayish involvement of the circumpapillary region. A large hemorrhage is present in the macula. The diagnosis was difficult and the angioid streaks are difficult to trace.

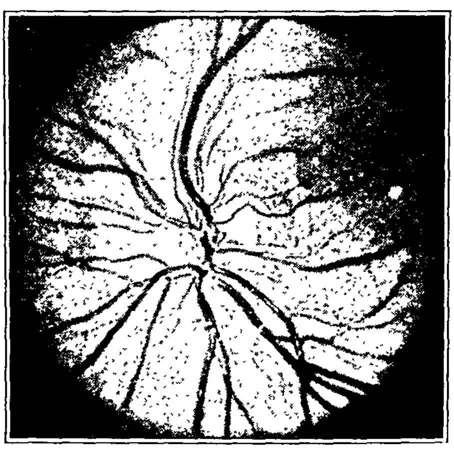

Fig. 8.—No diagnosis was made. There is circumpapillary involvement. The fundus is dark, the streaks are not typical and there is no pseudoxanthoma. The white beam under the disk is a reflection from the nerve layer of the retina.

disease. The presence of a myopic refraction cannot be taken into consideration, since angioid streaks may appear in a patient affected with myopia. Arteriosclerosis of the choroid, as a more or less independent choroidal disease, though generally affecting the posterior pole, may also involve the circumpapillary region (halo senilis). Since angioid

streaks have been mistaken for choroidal vessels, confusion is possible, but the characteristic course of preserved and sclerotic choroidal vessels will generally leave little doubt as to the correct diagnosis.

The identity of the early stages and varieties of angioid streaks may be difficult to establish. Tapetoretinal degeneration (Zeeman), also a choroidal disease, without any trace of angioid streaks (Batten) has been described. These anomalies will be dealt with more extensively later.

Since angioid streaks as such do not give rise to clinical symptoms, the patient is generally seen only if the macular region becomes involved. The whole group of possible macular conditions might therefore be mentioned as being of interest from the point of view of differential diagnosis. Since a serous exudate may be the first sign, angioneurotic, allergic and angiospastic conditions, macular degeneration and the first stage of Kuhnt's retinitis centralis atrophicans should be mentioned. The postedematous stage must be differentiated from macular degeneration, congenital anomalies, tumors and postinflammatory and traumatic conditions. However, a careful examination of the fundus and skin will nearly always lead to the correct diagnosis. Among these diseases, disciform degeneration is the most important and related. In both disciform macular degeneration (Junius and Kuhnt) and ordinary senile macular degeneration the serious changes around the disk, which are so characteristic in many cases of advanced angioid streaks, are often lacking. Angioid streaks is principally a circumpapillary disease, whereas disciform macular degeneration is a submacular disease.

This ophthalmoscopically related condition of disciform degeneration (fig. 9) of the macula (*scheibenförmige Entartung der Netzhautmitte* [Junius and Kuhnt [18]] ; *rétinite exsudative maculaire sénile* [Coppez and Danis [19]]) has been carefully studied histologically. In this disease a fibrous tissue develops between Bruch's membrane and the retina, appearing first under the pigmented epithelium. That the disease develops secondary to degeneration of Bruch's membrane, which, it is said, has a physiologic importance, was accepted as an established fact (Behr [20]; Rintelen [21]). Recently, however, on the basis of a careful study of the histologic picture in 3 private cases and a survey of the

18. Junius, P., und Kuhnt, H.: Die scheibenförmige Entartung der Netzhautmitte, Berlin, S. Karger, 1926.

19. Coppez, H., and Danis, M.: Rétinite exsudative maculaire sénile et rétinite circinée, Arch. d'opht. **43**:461, 1926.

20. Behr, C.: Ein weiterer Beitrag zur Anatomie und Pathogenese der scheibenförmigen Degeneration des hinteren Augenpols, Ztschr. f. Augenh. **75**: 216, 1931.

21. Rintelen, F.: Zur Histogenese des senilen submaculären Pseudotumors, Klin. Monatsbl. f. Augenh. **97**:673, 1936.

literature, covering 129 eyes, Verhoeff and Grossman [22] supported the view that the condition is primarily due to "some disturbance in the choriocapillaris, probably localized angiosclerosis." From the diseased choroid a hemorrhagic extravasate may settle between the pigmented epithelium and Bruch's membrane and undergo organization. This process is considered by Verhoeff and Grossman to be the "usual, if not the only, pathogenesis of this disease." Davenport [23] in his paper on senile macular exudative retinitis gave a clinical description of cases in which there was a large hemorrhage behind the macula. In the report of their third case Verhoeff and Grossman mentioned the presence of a serous exudate between the choroid and the retina, which is in agreement with Behr's findings; the latter expressed the belief that this exudate represents the first stage of the disease. Verhoeff and Grossman accepted this point of view as significant for cases of the juvenile type only and cases of serous retinitis secondary to "vascular disturbance dependent on some cause or causes yet to be determined."

Serous disciform chorioretinitis of the macula has been described by Batten,[24] Oguchi,[25] Kitahara [26] and Rauh [27] as a circumscribed macular detachment with no tendency to further development of a dialysis of the retina. Zeeman [28] demonstrated this condition in 7 patients; in 1 it followed iridectomy and in 1 there was a pit in the optic disk. Similar observations have been reported in cases of angioid streaks; recently Goedbloed described such a case. Though serous detachment of macula occurs in disciform degeneration, this simple fact is not sufficient reason for identifying this disease with retinitis disciformis. The diagnosis of juvenile disciform degeneration should be made only with great caution.

Typical for an earlier period of life is Coats's disease, retinitis exsudativa externa. In Coats's disease, Verhoeff, who personally made a histologic examination in several cases, also found new tissue present but not developing primarily beneath the pigmented epithelium. In the

22. Verhoeff, F. H., and Grossman, H. P.: Pathogenesis of Disciform Degeneration of the Macula, Arch. Ophth. **18**:561 (Oct.) 1937.

23. Davenport, R. C., cited by Rea, R. L.: Neuro-Ophthalmology, St. Louis, C. V. Mosby Company, 1938, p. 154.

24. Batten, R. W.: Macular Disease with Special Reference to Acute Primary Macular Disease, Tr. Ophth. Soc. U. Kingdom **41**:411, 1921.

25. Oguchi, C.: Eine besondere Form von Chorioretinitis centralis nebst Bemerkungen über nachbildartiges Skotom, Arch. f. Ophth. **110**:25, 1923.

26. Kitahara, S.: Ueber klinische Beobachtungen die der in Japan häufig vorkommenden Chorioretinitis centralis serosa, Klin. Monatsbl. f. Augenh. **97**:345, 1936.

27. Rauh: Ueber seltene Erkrankungen der Netzhautmitte, Ztschr. f. Augenh. **60**:209, 1926.

28. Zeeman, W. P. C.: Retinitis disciformis serosa, Nederl. tijdschr. v. geneesk. **81**:2995, 1937.

early stages a large amount of fibrin and hematogenous pigment is present. In the later stages cholesterol crystals surrounded by giant cells may be numerous. Verhoeff concluded that the origin of Coats's disease seems to be primarily retinal; that of Junius and Kuhnt's disease, primarily choroidal. In addition to these histologic differences, clinical anomalies of the vessels of the retina (fig. 10), convolutions of vessels and aneurysms are frequently seen in Coats's disease. Further, the condition is nearly always unilateral, whereas disciform degeneration is bilateral.

Though circinate retinitis has been reported in association with angioid streaks (also in a case of choroidal disease [Batten] in the brother of a patient with angioid streaks), this condition has to be considered as secondary to lesions in the deeper layers or to a related disease of

Figure 9

Figure 10

Fig. 9.—Disciform degeneration of the macula (Junius and Kuhnt). There is absence of angioid streaks and of circumpapillary involvement. The fundus is dark.

Fig. 10.—Coats's disease. Anomalies of the vessels are seen.

the retinal vessels (Morax [29]). This choroidal disease, as described by Batten,[30] will be discussed later.

TECHNIC OF HISTOLOGIC STUDY

The two eyes used for histologic study (1335 and 1335a) were enucleated post mortem; a small cap was cut off above and below and was preserved in a solution of formaldehyde. Serial pyroxylin sections of 15 microns were made and stained according to various methods or were preserved in alcohol with the caps. In the future it would be better to fix the specimens of one eye in a neutral solution

29. Morax, V.: Etude anatomo-pathologique d'un cas de dégénérescence circinée de la rétine, Ann. d'ocul. **163**:801, 1926.

30. Batten, D.: Angioid Streaks and Their Relation to a Form of Central Chorioidal Disease, Brit. J. Ophth. **15**:279, 1931.

of formaldehyde, since ordinary (acid) solution of formaldehyde may contain formic acid, which may cause small amounts of calcium and iron to disappear from the sections due to the formation of water-soluble calcium formate. In daylight formic acid develops continuously from solution of formaldehyde, so that the specimen should be kept in the dark. In addition, one eye should be preserved without the use of alcohol, so as to avoid solution of fatty material. Thus frozen sections should be made, either with or without embedding in gelatin.

GENERAL SURVEY OF HISTOLOGIC STUDY

The anomalies found in the two eyes were nearly identical, so that they will not be described separately. The eyes showed serious alterations around the disk, culminating in the macular region. In addition, an almost completely atrophic region of the retina was found at the nasal side of eye 1335a. Both of these areas in eye 1335a were easily located macroscopically, since here the elsewhere artificially detached retina was still attached to the underlying tissue. The retina was more adherent in eye 1335. In studying the sections stained with hematoxylin and eosin one was struck by the confusing variety of pathologic changes; there was, however, one constant and dominating feature in both eyes: the pathologic condition of Bruch's membrane (fig. 11 *A*). In a normal eye stained similarly the membrane was not visible, at least not with low magnification, whereas in these sections it seemed to be considerably thickened and was overstained with hematoxylin. Consequently, it was easily followed from the posterior pole to the equator, whereas toward the periphery it became less distinct. Besides the thickening and excessive basophilia, the membrane behind the equator showed a great number of defects, gathered especially around the disk. In the macular region the membrane was missing over considerable distances. Under high magnification the margins of most of the defects were as clearcut as though done with a knife (fig. 11 *B*). Therefore, it may be concluded that the defects were true ruptures of the membrane. The parts of the membrane bordering a defect showed no tendency to curl as does Descemet's membrane.

BRUCH'S MEMBRANE

Reconstruction of Defects in Bruch's Membrane.—A sufficiently accurate reconstruction of a plane view of Bruch's membrane in the nasal region adjacent to the disk was possible. On a projected picture of a section two parallel lines were drawn, one on each side of the optic nerve; a third line was constructed between these lines. A fourth line, nearly perpendicular to the first three, connected the end of Bruch's membrane to both sides of the optic nerve. It was assumed that the crossing point of lines 3 and 4 belonged to a line running perpendicular to the plane of the section (line 5). The distances between the edges

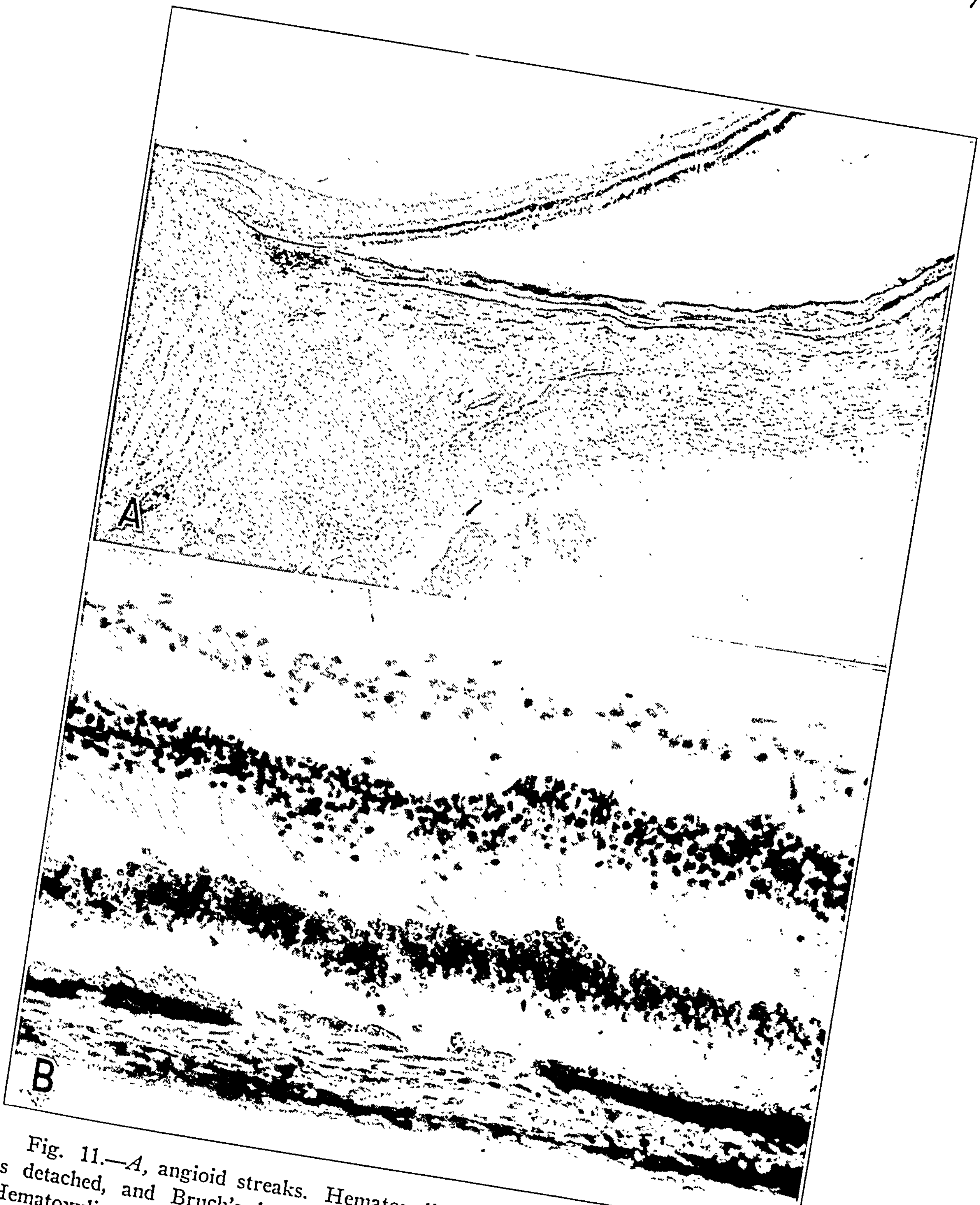

Fig. 11.—*A*, angioid streaks. Hematoxylin and eosin stain; × 40. The retina is detached, and Bruch's layer is thickened and broken. *B*, angioid streaks. Hematoxylin and eosin stain; × 225. Note the clearcut edges of rupture in Bruch's layer.

of several ruptures to the corresponding crossing points were measured in a number of subsequent sections and transcribed to a schema of parallel lines, so spaced as to represent adequately the subsequent sections. It is true that line 5 is not in reality a straight line, but the including of many ruptures in these estimations acted as a valuable correction. By comparing the transcribed dots of two adjacent lines in the reconstruction schema, it became immediately obvious that a slight correction, either to the right or to the left, was necessary. Figure 12 demonstrates the similarity of the reconstructed defects to the clinical picture of angioid streaks. They are different from ruptures of Bruch's membrane in cases of high myopia.

Extent of Involvement of Bruch's Membrane.—In the sections studied the thickening and basophilia of the membrane proved to be constant and complete in the region behind the equator. In the macular region the membrane was sometimes atrophic. Apparently normal areas, which

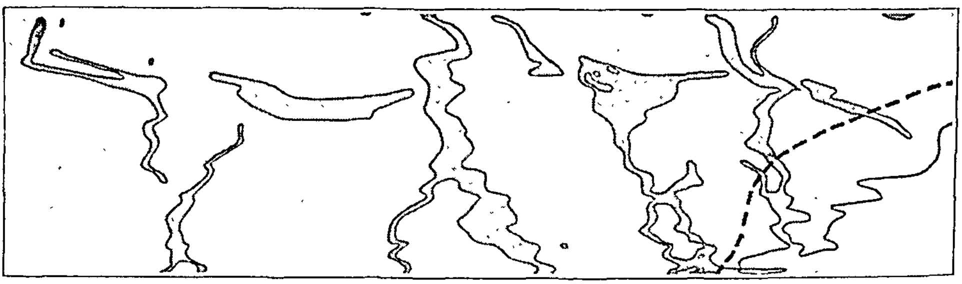

Fig. 12.—Angioid streaks. Reconstruction of ruptures in Bruch's layer from the sections. The dotted line represents the projected circumference of the optic nerve.

did not stain with hematoxylin, appeared toward the periphery. The transition to these areas was short, though not abrupt. Thus normal areas alternated with thickened and overstained areas over the entire periphery till the pars plana was reached. It was practically impossible to make a plane view reconstruction of the diseased parts in this region, except for two areas at the extreme periphery, where an ordinary cystic degeneration of the retina served as a landmark in localizing the diseased portion (fig. 13), so that a reasonably accurate reconstruction was possible. Thus it could be proved that the two affected areas were small, nearly round islands of diseased membrane.

The caps cut from the eyes before embedding included a part of the choroid.

An effort to study Bruch's layer in plane view in small pieces of depigmented choroid, stained with orcein and made translucent after the procedure that was employed by Spalteholz, was made. Knowledge beforehand that this degeneration existed made it possible to trace

the affected areas, in which the fibers were somewhat coarser, but the results were disappointing; it was not possible to draw any conclusions whatever from these pictures alone. Perhaps this was due to the fact that the material available for these investigations was limited. Other pieces of choroid were stained with hematoxylin. The irregular distribution of the diseased areas could be observed. However, it was impossible to make a clear photograph, owing to the heavily pigmented epithelium on the one side and of the stained choroid on the scleral side. Therefore, a drawing was made to demonstrate these areas (fig. 14). For better results it seemed reasonable to depigment the specimen before staining with hematoxylin. This procedure spoiled the basophilia of Bruch's membrane, so that the results were unsatisfactory. For this

Fig. 13.—Angioid streaks. Hematoxylin and eosin stain; × 225. Cystic degeneration of the peripheral portion of the retina is present. Bruch's layer is scarcely visible and is thickened in the region under the smaller cyst.

reason figure 14 is of a hematoxylin-stained section which has not been depigmented.

The broken ends of the membrane and the membrane itself bordering the ruptures were straight, not undulated, so that the degeneration seemed to be associated with a process of shortening, which, however, as could be seen from the reconstruction, was not as marked as might appear from a study of sections. A detailed study of Bruch's membrane requires knowledge of its normal structure.

Normal Histologic Structure of Bruch's Membrane.—Wolfrum described Bruch's membrane as consisting of two layers, an outer collagenous elastic layer, measuring about 0.5 micron, and a much thinner

inner layer of no measurable thickness, less than 0.25 micron, the latter being the basal layer of the pigmented epithelium of the retina.

Wolfrum [31] (and former authors) described the very delicate elastic fibers of the elastic membrane as often making hairpin turns to enter the choriocapillaris, where they become considerably thickened. In eye

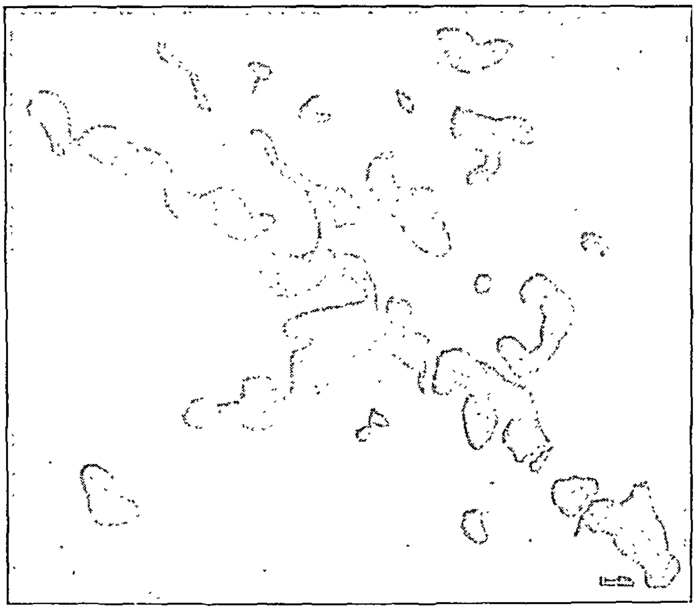

Fig. 14.—Drawing showing areas of degeneration in Bruch's layer. A piece of choroid was stained in toto with hematoxylin. There was a macular staining of Bruch's layer which could not be photographed, since the pigmented epithelium veiled the picture. Depigmentation spoiled the hematoxylin stain of Bruch's layer.

Fig. 15.—Hypertension. Hematoxylin and eosin stain; × 950. Septums in the choriocapillary layer are inseparably connected with Bruch's layer.

P. A. 1293 (a case of hypertension) the relation of Bruch's layer to the septums in the choriocapillaris was pathologically exaggerated (fig. 15); according to Wolfrum this layer must be considered not as a membrane,

31. Wolfrum, M.: Beiträge zur Anatomie und Histologie der Aderhaut beim Menschen und bei höheren Wirbeltieren, Arch. f. Ophth. **67**:307, 1908.

but as the extreme inner region of the elastic system of the choroid. These statements could be confirmed in sections of sarcomatous eyes. The outer layer is not an elastic "membrane" but a network of tiny elastic and collagenous fibers. Whether it functions as a membrane cannot be judged. It should be called "Bruch's layer." There can be no historical objection to connecting Bruch's name with this layer, since this author observed the thick posterior layer only and was not acquainted with the minute histologic structure of the region. (The original article [32] was not available to me.) There exists a potential space between the inner and the outer layer, and normally they are never adherent, so that leukocytes may penetrate between them. The inner layer is always adherent to the pigmented epithelium. It should be called the basal membrane of the pigmented epithelium. The name Bruch's membrane should be maintained in all those instances in which a separate study of the two layers has proved impossible. This is the case in nearly every reported study in which a description of the condition of Bruch's membrane is given.

In the following discussion of Bruch's membrane consideration will be given to whether the membrane has degenerated as a whole or whether only one of the two layers has been affected.

Bruch's Membrane in Pyroxylin Sections.—The thickening and basophilia in the sections stained with hematoxylin have been described (fig. 17 *C*). Since thickening, basophilia and brittleness of the elastic tissue are also found in pseudoxanthoma, these conditions might be considered identical, a supposition which raised the theoretic question of a disease of the elastic system as a whole. Such a satisfactory explanation was apparently upset, however, by evidence found in sections stained with orcein, a dye staining electively elastic fibers; such sections were compared with the normal membrane of a sarcomatous eye. At the posterior pole the membrane stained well but was not thicker than normal (fig. 16 *A*). Toward the periphery it became somewhat thicker, without, however, decidedly differing from the membrane of the sarcomatous eye. It was impossible to recognize the islands of degeneration with this stain. In front of the thin elastic layer a thick, homogeneous, faintly staining layer was seen, which by comparison with the sections stained with hematoxylin could be interpreted as being the thickened basal membrane of the pigmented epithelium (fig. 16 *A*). Combined staining with hematoxylin and orcein did not furnish satisfactory results, since orcein interfered with the hematoxylin. At first it was concluded that the basal membrane was thickened and pathologic, Bruch's layer being fairly

32. Bruch, C.: Untersuchungen zur Kenntnis des körnigen Pigments der Wirbelthiere in physiologischer und pathologischer Hinsicht, Zurich, Meyer u. Zeller, 1844.

Fig. 16.—*A*, angioid streaks. Orcein stain; × 810. In the upper part of *A* Bruch's layer is thin. Apparently anterior to it is a thick amorphous layer. In the lower part are seen the third stage of degeneration of the choroidal vessel and atrophy of the elastic tissue. *B*, angioid streaks. Hematoxylin and eosin stain; × 580. Bruch's layer in the macular region is homogeneous and crisp.

normal. But on further study, especially of peripheral and damaged sections stained with hematoxylin, it was revealed that the membrane gave a false impression of being thickened. One of the sections in which an apparently thick, darkly stained membrane was seen was studied further under powerful blue filtered illumination. The membrane was then seen as being delicately stained and translucent. Its cut surface could be easily distinguished from the shadow-like appearance of that part of the membrane which was not in focus. By bringing all parts of the membrane in and out of focus, there remained not the least doubt that whereas under ordinary light the membrane stained with hematoxylin appeared thick, it was in reality just as thin as that seen in the sections considerably less intensely stained with orcein. It may be concluded that the apparent considerable thickening of the membrane in sections stained with hematoxylin was, for the greater part, an optical illusion, the overstaining making it impossible to distinguish the plane of section from the shadow of that part of the membrane out of focus. The membrane at the degenerated areas in the periphery did indeed seem to be somewhat thickened, whereas at the posterior pole it was decidedly thinner than normal, so thin in fact that at the macular region it resembled a thin sheet of crisp old paint which had peeled off (fig. 16 *B*). It may be concluded that the onset of degeneration as seen at the periphery causes a slight thickening of Bruch's membrane, which, as the degeneration proceeds, becomes thinner, till at the posterior pole it may become atrophic. For a more detailed study it is necessary to consider the behavior of the pigmented epithelium over the diseased membrane.

Pigmented Epithelium and Involvement of Bruch's Membrane.— Since the pigmented cells are inseparably connected with their basal layer, the so-called inner layer of Bruch's membrane, they have to be included in a study of this membrane.

It is interesting to consider a possible relation between the development of an amorphous layer, probably an exudate, and the degenerated areas at the periphery of Bruch's layer, since these exudates occurred at the site of the degeneration (fig. 17 *A* and *B*). That the pigmented epithelium was as a whole pushed aside by this exudate points to the fact that its basal membrane must have been unimpaired. It did not stain with hematoxylin as did Bruch's layer, from which it was separated by the exudate. Therefore, it may be concluded that the basal membrane of the pigmented epithelium does not share in the degeneration of Bruch's layer, at least not in the feature of becoming excessively basophilic.

Evidently the degeneration of Bruch's layer allows a passage of fluid from the choriocapillaris, or it induces the pigmented epithelium

to secrete a subepithelial mass, in the manner described by Koyanagi [33] in hypertension. The latter supposition is the less probable since as degeneration proceeds, which is the case as one passes from the periphery toward the equator, the exudate is missing and the pigmented epithelium lies directly on the continuously degenerated Bruch's layer (fig. 17 *C*). Furthermore, the secretory activity of the pigmented epithelium described by this Japanese author is considerably different from that considered here. As one approaches the posterior pole, ruptures bridged by apparently unaltered pigmented epithelium may be seen—a fact which confirms the supposition that the basal membrane behaves independently and does not share in the degeneration. Other

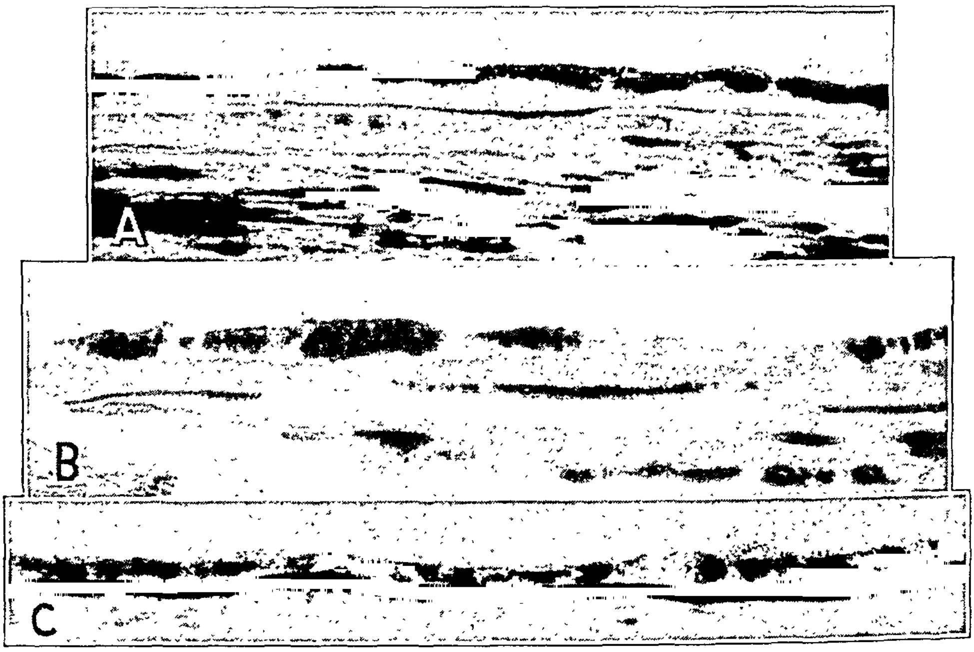

Fig. 17.—*A* and *B*, angioid streaks; peripheral region. *A*, pyroxylin section. Hematoxylin and eosin stain; × 590. The exudate at the site of the localized degeneration of Bruch's layer is pushing off the pigmented epithelium. *B*, paraffin section. Hematoxylin and eosin; × 1,050. Note the isolated degeneration of fibers in Bruch's layer. There is an exudate or a homogeneous layer at the site of the degeneration between the pigmented epithelium and Bruch's layer. *C*, angioid streaks; equatorial region. Hematoxylin and eosin stain; × 480. Bruch's layer is considerably "thickened." The pigmented epithelium is apparently fairly normal and is seen lying directly on Bruch's layer.

serious changes which occur nearer the posterior pole will be described later.

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33. Koyanagi, Y., and Kinukawa, C.: Gleichartige Veränderungen des retinalen Pigmentepithels und des renalen Tubulusepithels bei verschiedenen Vergiftungen (Beiträge zur Pathogenese der Retinitis albuminurica), Arch. f. Ophth. **137**:261, 1937.

Minute Anatomy of the Degeneration of Bruch's Layer.— Even in the sections embedded in pyroxylin and stained with orcein, the individual fibers still could be easily demonstrated (fig. 18 *A*) if the membrane were twisted. The fibers are more delicate and regular in a normal

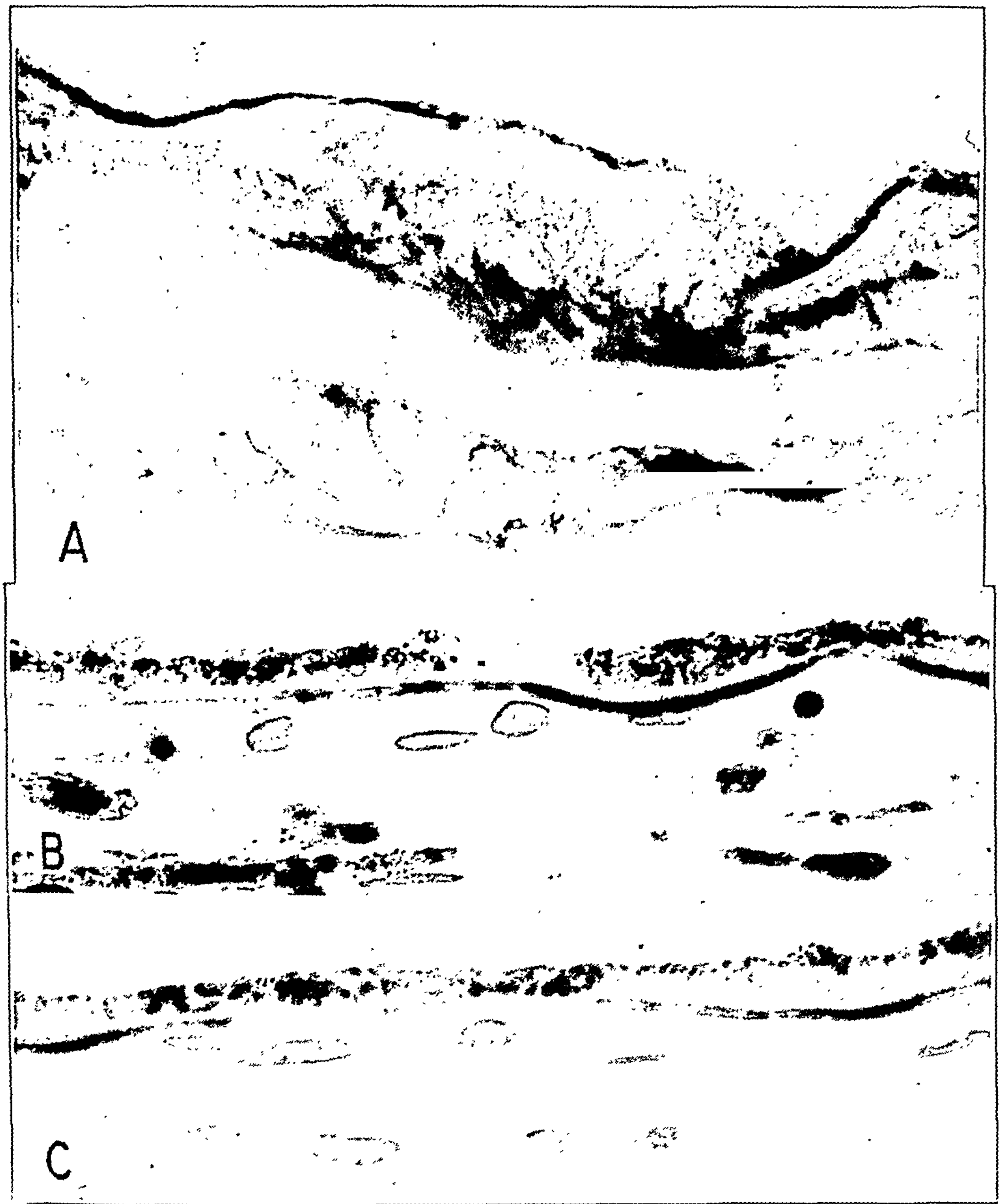

Fig. 18.—*A*, angioid streaks. Damaged pyroxylin section. Orcein stain; × 800. Bruch's layer is twisted. Individual fibers are traceable but irregular and coarser. The fibrillar character of the membrane is still maintained. *B* and *C*, angioid streaks. Paraffin sections. Hematoxylin and eosin stain; × 1,050. There is degeneration of individual fibers at the borders of the islands of complete degeneration.

eye (fig. 43). The aforementioned caps which were cut from the eyes included a part of the choroid; this was carefully separated from the sclera and paraffin sections made of it. In the sections stained with hematoxylin it could be definitely proved that basophilia of Bruch's layer does not at the onset occur as a whole but that isolated fibers within the layer degenerate individually. The fibers seen in figure 18 *B* and *C*, though cut, are still viewed partly in plane view, owing to the fact that the thickness of the sections is 6 microns and the thickness of Bruch's layer, from 0.5 to 1 micron. Degenerated fibers, though occasionally seen in an apparently normal part of the layer, are generally observed in the neighborhood of more completely degenerated regions. It appears that the layer in the more severely affected areas loses its fibrillar character. Becoming more membranous, it stains more evenly, showing only a faint network of more or less sharply marked off degenerated fibers. The disappearance of the individual fibers was excellently demonstrated in the pyroxylin sections (fig. 19 *A*), if the layer was twisted and the pigmented epithelium torn off. Finally, no trace of individual fibers is left (fig. 16 *B*). The affinity of Bruch's layer for orcein is maintained until complete atrophy develops (fig. 19 *B*).

Special attention was paid to the behavior of the elastic fiber as it dips into the choroid. As a rule this part of the fiber remained apparently normal, but occasionally one could be seen which stained a faint blue. Small lines and buds of different shape were often seen at the outer surface of the layer, some of which, under binocular examination, proved to be ruptured pieces of the layer itself. It may be concluded that basophilia of that part of the elastic fibers of Bruch's layer which dips into the choroid does occur, but is exceptional and slight.

The intense staining of the layer with hematoxylin suggested the possibility of the presence of calcification. The membrane was tested for the presence of calcium, iron, lipoids and amyloid.

The membrane stained by the Kossa method apparently stained faintly (calcium test), more deeply at the posterior pole. It did not, however, stain so intensely as with hematoxylin, and when one remembers that an unstained membrane under an ordinary microscope, but especially a microscope with a narrow diaphragm, can give the appearance of being stained, no definite conclusion is possible. Perhaps the use of ordinary (acid) solution of formaldehyde caused a disappearance of the calcium. The more this possibility was considered the more it seemed probable, since metaplastic bone formed in the eye did not give rise to difficulties in making the sections, though they were not decalcified. Moreover, this

bone did not give a positive reaction to the Kossa test. Control tests with identical results were performed in the pathologic laboratory.[34]

With the iron test for blood pigments it was accidentally found that Bruch's membrane stained intensely, so that there was a pathologic amount of iron in this layer, whereas the rest of the eye was free from blood pigment (fig. 45 *A*). Utmost care and comparative staining excluded the possibility of the iron being due to artefact. In stained

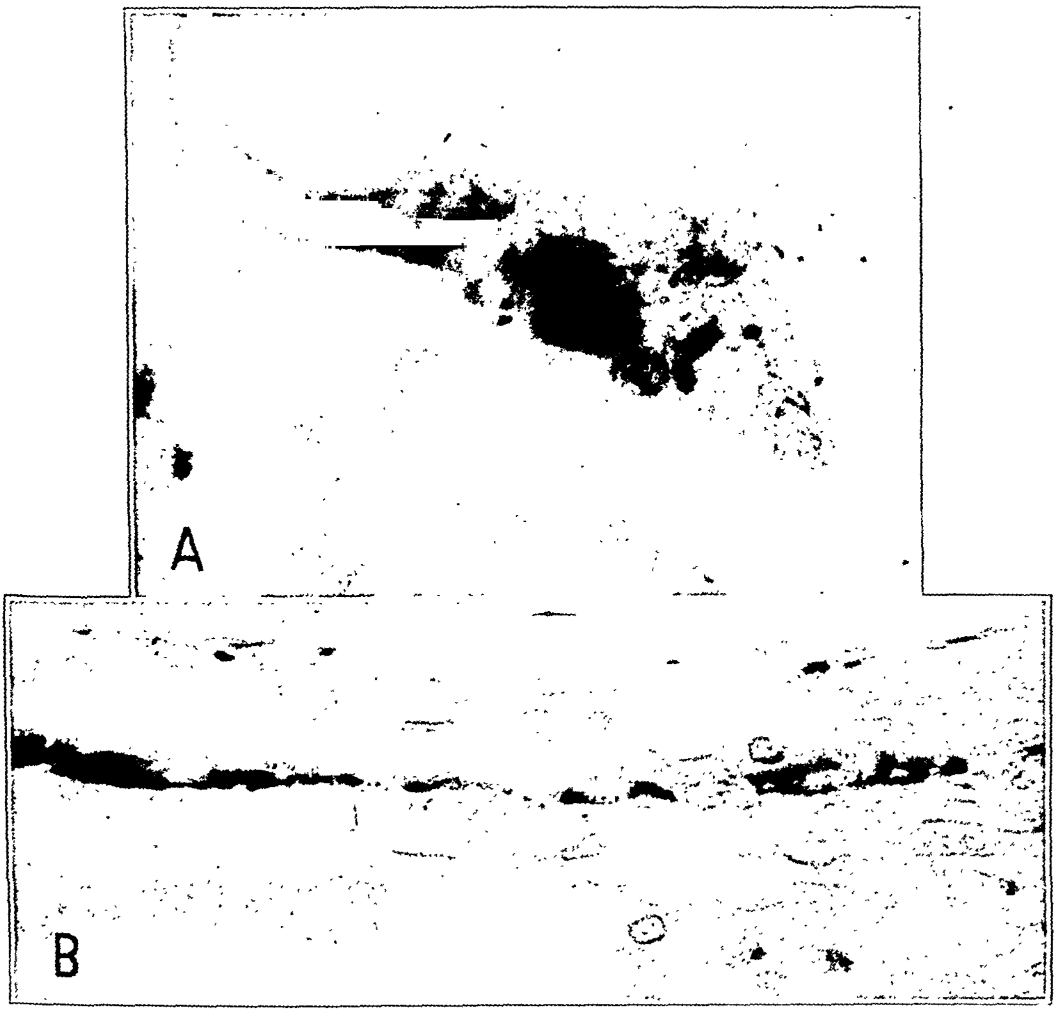

Fig. 19.—*A*, angioid streaks. Damaged pyroxylin section. Hematoxylin and eosin stain; × 1,050. Bruch's layer is twisted and broken and stained with hematoxylin. The fibrillar character is traceable but indistinct. *B*, angioid streaks; macular region. Depigmented specimen. Orcein stain; × 1,050. The last rests of the disappearing Bruch's layer still stain with orcein.

sections of various eyes Bruch's layer was found to give a negative reaction to the iron test. These control sections had been pre-

34. Böck reported the presence of lime but did not perform the Kossa test (Gefässähnliche Netzhautstreifen [Angioid Streaks] und Pseudoxanthoma elasticum, Ztschr. f. Augenh. **86**:238, 1935).

viously handled and preserved in the same way. The possibility exists that Bruch's layer contained calcium in vivo, in connection with which iron was precipitated, since iron has a tendency to precipitate in, or in the neighborhood of, calcified regions. Hueck (according to Schupisser [35]) was the first to demonstrate the affinity of lime for the iron in preserving and staining fluids. But no calcium was present in the sections used for the present study at the time of staining. The results of the test for the presence of iron in the neighborhood of the metaplastic bone were completely negative. This makes it probable that the presence of this iron is independent of a primary presence of calcium.

There may be another source for the presence of iron in Bruch's membrane. It is known that in angioid streaks recurrent smaller and larger hemorrhages may occur. Therefore, the iron in Bruch's membrane might be considered as having originated from former hemorrhages, the blood pigment having completely disappeared from the choroid and retina, whereas it was preserved in the membrane. This, however, cannot be the case, since just the reverse situation was found in siderotic eyes, stained for comparison. It was shown that the pigmented epithelium has an affinity for iron but that Bruch's membrane has not. It remained unstained in eyes with siderosis and hemorrhage, with abundant iron pigment. This finding is not in accordance with Ehrlich's statement (according to and confirmed by Schupisser) that elastic fibers in the neighborhood of hemorrhages have a tendency to become loaded with iron salts. The behavior of pseudoxanthomatous skin to the iron test seemed interesting. I succeeded in obtaining only a piece of pseudoxanthomatous skin, which had been preserved in a solution of formaldehyde for three years. All of the degenerated elastic fibers gave a negative reaction to the Kossa test (calcium); a certain area in the degenerated elastic tissue stained with hematoxylin and was likewise positive to the iron test. It would seem likely, therefore, that siderosis precedes the calcification of the elastic fibers. It is not probable that the iron may have originated from a preceding hemorrhage. I could not find a statement as to whether elastic fibers loaded with iron salts stain with hematoxylin elsewhere. The membrane stained with the Kulschitzky stain, a myelin sheath stain, so that probably lipoids not soluble in alcohol were present, though it must be admitted that this test is not absolutely reliable. Nevertheless, this fact is interesting, since from reports in the literature the appearance of iron, when independent and not secondary to calcium, is always associated with the presence of myelin-like substances (Schupisser). This again supports

35. Schupisser, H.: Ueber Eiseninkrustation der Bindegewebssubstanzen, Virchows Arch. f. path. Anat. **239**:320, 1922.

the view that the presence of iron is secondary not to dissolved but to originally present lime in the elastic fibers. It is regrettable that the foregoing statements cannot be made with certainty, since any calcium present may have been dissolved. They emphasize, however, the importance of handling the material in such a way that any solution of calcium or contamination with iron is prevented. It seems probable, though with some restrictions, that iron plays a role in the process of angioid streaks, although hemochromatosis has not been reported.[36]

There was no amyloid in the membrane or in the other parts of the eye.

Specificity of Involvement of Bruch's Layer.—In studying the collections of specimens showing involvement of Bruch's membrane in the laboratory at the University of Amsterdam, I found a similar basophilia (eye P. A. 980, fig. 20). The eye was enucleated from an old man aged 82 because of pain following an unsuccessful operation for cataract. The posterior segment of the eye was free from inflammation. Bruch's layer stained as it does in angioid streaks, though not so dark. It was impossible to make a photograph similar to that shown in figure 11 *A* on account of the intense pigmentation around the disk. Small patches of the membrane at some distance from the disk had degenerated, but at the equator the membrane was normal. The islands of degeneration resembled those found at the periphery in angioid streaks. It is probable, however, that there is a difference between the degeneration of angioid streaks and that of senility, since in this eye degeneration in the moderately affected areas was more dustlike and individual fibers could not be traced in the sections stained with hematoxylin. Figure 20 shows this degeneration in an area in which pigmentation was less intense. The iron test was slightly positive in the affected areas, the membrane staining a faint blue. This eye further demonstrated the fact that a considerable degeneration of Bruch's layer does not necessarily cause a disciform degeneration of the macula. Ruptures of the membrane were not found.

In eye P. A. 1293 the layer of Bruch stained faintly and homogeneously with hematoxylin. The course of the fibers between the chorio-

36. The iron test was negative in Böck's case; however, the eyes were fixed in Zenker's and Orth's fluid. According to Schmorl, these fluids cannot be used if iron tests should be performed. The Turnbull blue test (after Firmann and Schmelzer) was performed with the specimen studied by me, whereas Böck used the *Berlinerblau* reaction, which is less reliable (*weniger genau* [Romeis], *am wenigsten zuverlässig* [Schmorl]). (See Romeis, B.: Taschenbuch der mikroskopischen Technik, ed. 13, Berlin, R. Oldenbourg, 1932, p. 341. Schmorl, G.: Die pathologisch-histologischen Untersuchungsmethoden, ed. 14, Leipzig, F. C. W. Vogel, 1925.)

capillary layers was easily traceable, since they stained slightly. The specimen was removed from a patient with vascular disease associated with a pale cupped disk and a glaucoma-like field.

The condition which most resembles the degeneration of Bruch's membrane in angioid streaks is senile degeneration of the membrane. Though the differences are notable, still the same has been said by other authors of pseudoxanthoma in relation to senile degeneration of the skin.

The degeneration of the membrane which occurs in disciform degeneration is regarded as the basis of this disease by Behr and Rintelen,

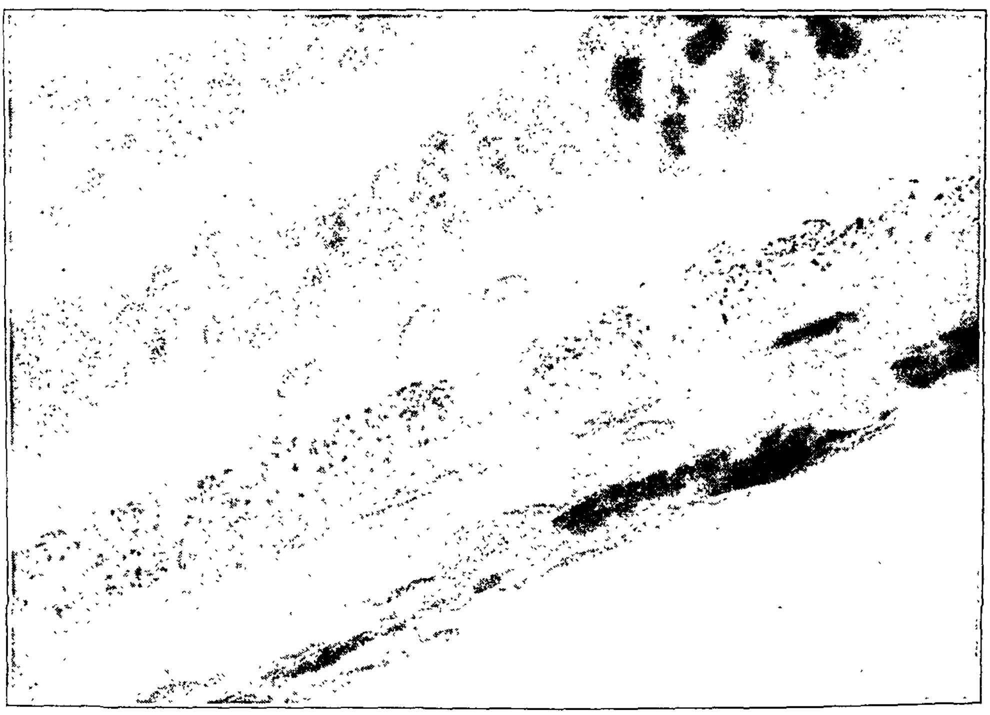

Fig. 20.—Senile degeneration of Bruch's layer. Hematoxylin and eosin stain; × 800. There is dustlike degeneration in the less affected areas.

whereas, on the other hand, Verhoeff expressed astonishment at finding the membrane relatively so well preserved. Doubling of the membrane, as is described in disciform degeneration, was in my sections an optical illusion caused by twisting of the membrane. The affinity of the membrane for orcein was often lost in the reported cases of disciform degeneration, whereas in angioid streaks it is constant. The peripheral parts of the membrane were normal.

Ruptures were present, but small and few in number. Verhoeff expressed the belief that the ruptures are secondary to the activity of the mesodermic cells and stated that "proliferation of connective tissue cells without causing breaks could be definitely recognized." In one of his cases the membrane was broken at but one point, the site of a

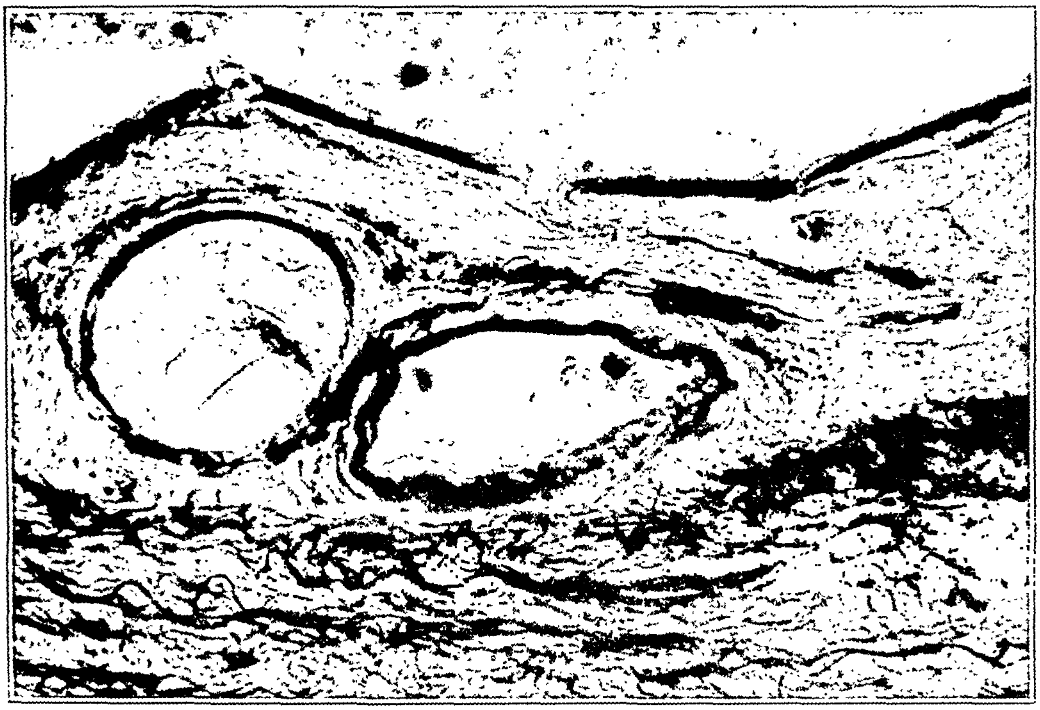

Fig. 21.—Choroidal arteriosclerosis. Orcein stain: × 325. There are ruptures of Bruch's layer showing torn fibrillar borders, arteriosclerotic arteries and degeneration of the elastic tissue of the choroid.

Fig. 22.—Myopia and ruptures of Bruch's layer. The ruptures are closed by cicatricial tissue (white streaks). Note the translucency of Bruch's layer and the visibility of the choroidal vessels.

severe hemorrhage. A good picture of basophilia and rupture in disciform degeneration was given by Wölfflin.[36a] Ruptures of Bruch's layer are likely to occur under various circumstances. Blunt trauma to the eye may cause the membrane to rupture, leaving the retina unimpaired. It is therefore not impossible that it may be found broken in the presence of inflammation. I found a break of the layer in the periphery of a sarcomatous eye, probably traumatic, without reactions of the underlying choroid. Ruptures were found in eye P. A. 1129, which was affected with albuminuric retinitis and choroidal arteriosclerosis (fig. 21); the membrane was not basophilic and looked more torn than broken. High myopia alone produces ruptures which may be compared with those found in angioid streaks. They are seen, however, only in the region of the posterior pole, where their occurrence can be easily explained by the distention of the wall of the eye. Moreover, the membrane is not basophilic, and their form as well as their localization differs from that found in angioid streaks. They are seen, however, only in the region of thickened in angioid streaks. The ruptures are frequently closed by an *Ersatzmembran,* a substitute membrane which gives them the white aspect seen clinically (fig. 22). Nevertheless, it cannot be denied that ruptures occur in myopia which resemble clinically those seen in angioid streaks. On this similarity Kofler[37] founded his theory that angioid streaks are ruptures of Bruch's membrane.

It may be concluded that the described type of degeneration of Bruch's layer is specific for angioid streaks.

(To be concluded)

36a. Wöfflin, E.: Beitrag zur pathologischen Anatomie der Retinitis exsudativa externa, Arch. f. Ophth. **117**:33, 1926.

37. Kofler, A.: Beiträge zur Kenntnis der Angioid Streaks, Arch. f. Augenh. **82**:134, 1917.

CIRCULATORY DISTURBANCES IN RETINA IN ARTERIOSCLEROSIS AND IN ESSENTIAL ARTERIAL HYPERTENSION

HERMAN ELWYN, M.D.
NEW YORK

This review is an attempt to correlate the clinical and ophthalmoscopic signs of circulatory disturbances in the retina in arteriosclerosis and in essential arterial hypertension with certain fundamental pathologic elements which underly local circulatory disturbances. These elements are the ones usually discussed in treatises on general pathology under the heading of local circulatory disturbances. I have selected such material from these treatises as is needed for my purpose. Complete discussions are found in standard works, such as those by Aschoff,[1] Hueck,[2] Ricker [3] and Dietrich.[4]

LOCAL CIRCULATORY DISTURBANCES IN AN ORGAN

The circulation of an organ forms a part of the general circulation and is dependent on the general arterial pressure and on the general venous return flow. It also constitutes an independent unit in the service of the particular organ, independent in a way of the general arterial pressure and the general venous return flow and dependent on the needs and activities of the organ at a given time. The arteries, capillaries and veins which constitute the circulatory units are under the control of the vasomotor system, which regulates the degree of constriction and dilatation of the vessels. The local circulatory apparatus can therefore be subject to two kinds of disturbances: (1) vasomotor disturbances, which result from a change in the neural control, and with neural control I shall tacitly include hormonal control, and (2) mechanical disturbances, such as those produced by mechanical obstruction of the lumen of the vessel either by a thrombus or by an embolus within the lumen, by changes in the wall of the vessel or by compression from without.

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1. Aschoff, L.: Pathologische Anatomie, ed. 7, Jena, Gustav Fischer, 1928, vol. 1, p. 461.

2. Hueck, W.: Morphologische Pathologie, Leipzig, Georg Thieme, 1937.

3. Ricker, G.: Pathologie als Naturwissenschaft, Berlin, Julius Springer, 1924.

4. Dietrich, A.: Allgemeine Pathologie und pathologische Anatomie, Leipzig, S. Hirzel, 1933, vol. 1, p. 109.

VASOMOTOR DISTURBANCES

The effect of stimuli on the vasomotor nerves has been shown by Ricker [3] to be as follows: Weak stimuli affect the dilator fibers, causing dilatation of the vessels with an increase in the blood flow. The constrictor fibers are not affected and remain irritable. Slightly stronger stimuli affect the constrictor fibers, causing constriction of the arteries with a slowing of the circulation in the capillaries and veins. Strong stimuli cause paralysis of the constrictor fibers and stimulation of the dilator fibers. This produces at first a dilatation of the vessels and an increase in the blood flow. But the dilator fibers are soon paralyzed, and above the dilated vessel the artery is constricted. The result is a slowing of the blood flow to complete cessation or stasis.

Depending on the action of agents on the vasomotor nerves, there can be distinguished: (1) simple, or active, hyperemia, with an increase in the blood flow (congestive hyperemia), (2) hyperemia, with a slowing of the blood flow, or (3) stasis or cessation of the blood flow.

Simple, or Active, Hyperemia.—Simple, or active, hyperemia is produced by stimulation of the dilator fibers, the constrictor fibers remaining unaffected. Arteries, capillaries and veins are dilated, and the blood flow is increased; there is also an enormous increase in the number of open capillaries. This form of hyperemia is found in a functionally active organ, such as a contracting muscle or a secreting gland. The hyperemia is temporary during activity, and there is only a slight loss of fluid through the capillary wall. Related to this form of hyperemia is the compensatory hyperemia which occurs in the remaining one of a paired organ after the loss of the other; also the collateral hyperemia which occurs in the neighborhood of an infarcted area.

Hyperemia with Slowing of the Blood Flow; Peristatic and Prestatic Hyperemia.—Peristatic and prestatic hyperemia occur with a stimulation of the dilator fibers and a change in the constrictor fibers, which are either diminished in activity or are completely inactive. In the area of the inactive constrictor fibers the arteries, capillaries and veins are dilated, and the circulation is slow and irregular; central to this area the artery is constricted. This form of hyperemia Ricker calls peristatic hyperemia. This classification includes neuroparalytic hyperemia and inflammatory hyperemia. Characteristic for this form is the increased passage of fluid through the capillary walls and its accumulation in the surrounding tissues.

When the artery central to the affected area is still more constricted, there is a still greater dilatation of the vessels in the affected area and a still greater slowing of the blood stream. Ricker calls this form prestatic hyperemia. Circulation is still present but slowed. This form is characterized by the passage of red blood corpuscles through the capillary

walls. They pass through the capillary wall singly at one point, or hemorrhages occur at a number of points.

The peristatic and prestatic forms of hyperemia are of great importance in that they explain the occurrence of edema and hemorrhages.

Stasis.—When the artery central to the affected area is markedly narrowed or completely occluded, the activity of the vasomotor fibers in the affected area is completely lost, the vessels are relaxed and circulation is at a standstill. The capillaries are filled with a column of red corpuscles packed closely to such a degree that the individual corpuscles cannot be recognized. The blood plasma is already lost, having passed into the veins or through the walls of the capillaries into the tissues. The mass of red corpuscles is not agglutinated or thrombosed but simply packed together. With the cessation of the central arterial constriction, the red corpuscles in the static capillary become separated from one another and circulation is resumed. Diapedesis does not occur during the period of stasis, as all movement of red corpuscles has ceased. With long-continued stasis, the tissues supplied by the vessel undergo necrosis.

MECHANICAL DISTURBANCES

The circulation of an organ can be disturbed mechanically by an obstruction to the flow of blood in the arteries or in the veins. Obstruction results (*a*) from compression of the vessel from without, as by an exudate or a growth, or (*b*) from changes within the vessel, such as arteriosclerosis, endarteritis obliterans, thrombosis and embolism.

Complete obstruction of an artery occurring suddenly is followed by a reflex constriction of the vessel peripherally to the point of obstruction and by ischemia of the area supplied by the artery. Soon there occurs a dilatation of the capillaries and venules distal to the constricted area, and, as in vasomotor constriction, there is produced a state of peristasis. From the plasma which is still present in the dilated vessels distal to the constriction or has accumulated there by the backflow from the veins, fluid transudes into the surrounding tissue. When the obstructed vessel is an end artery, such a complete obstruction is followed by an anemic infarct. When the obstruction is not complete, the varying degrees of reflex constriction below the point of obstruction and of dilatation of arterioles, capillaries and venules distal to the constriction produce the conditions of peristasis and prestasis; with the latter, there occur transudation of fluid and hemorrhages to a varying degree.

Obstruction of a vein is followed by cessation of circulation in the vein and by a reflex constriction of the supplying artery. Before circulation stops, there is first a slowing of the blood flow, producing a state

of prestasis with its attendant hemorrhages. Soon a condition of stasis is reached. This type of hyperemia is spoken of as stasis hyperemia.

SUMMARY

Local circulatory disturbances in an organ, whether due to vasomotor changes or to mechanical obstruction, result in various degrees of constriction and of dilatation of the terminal units, leading to states of active hyperemia, peristasis, prestasis, ischemia or obstruction anemia and their consequences.

IMMEDIATE RESULTS OF LOCAL CIRCULATORY DISTURBANCES

Simple, or active, hyperemia is a transitory condition and without any appreciable consequences. Peristasis is accompanied by the passage of fluid through the capillary walls and passes quickly into prestasis, in which hemorrhages occur. Stasis is accompanied by hemorrhage in the prestatic marginal areas, and obstruction anemia is followed by infarction of the involved area. The immediate results of local circulatory disturbances are, therefore, (1) transudation of plasma into the tissues, (2) hemorrhage and (3) infarction.

Transudation of Fluid.—Under normal conditions the passage of fluid through the capillary wall is regulated by the vegetative nervous system in accordance with the needs of the tissues. Hueck [2] has demonstrated that the protoplasm of the endothelial cells of the capillaries and of the basement membrane is capable of undergoing a transitory liquefaction to a degree which permits numerous openings to form which allow the plasma to pass through and, in case of larger openings, also the red corpuscles. In states of peristasis and prestasis the slowing of the circulation in the capillaries causes a diminution in the supply of oxygen and nutrition to the walls of the capillaries and creates the conditions which permit the liquefaction of the protoplasm of the endothelial cells and of the basement membrane. The fluid which transudes is blood plasma and contains a considerable amount of protein; fibrin is then deposited in the tissues.

Hemorrhage.—Hemorrhage as a result of local circulatory disturbances always occurs by diapedesis and practically always occurs through the capillary walls. According to Hueck, such a passage presupposes, as in the case of the passage of fluid, a liquefaction of the protoplasm to a degree which permits openings to form at numerous points. These openings last only for a few moments. They may be close together and in their aggregate permit the formation of large hemorrhages. The immediate cause of such hemorrhages by diapedesis is always a condition of prestasis.

Infarction.—With complete obstruction of an end artery, the tissues supplied by it do not receive any oxygen or nutrition. Death of the tissues occurs at a varying time, depending on the character of the tissue. In the brain it occurs within a few minutes after the obstruction. In the retina it is said to occur after an obstruction of about thirty minutes; in the kidney, after an obstruction of about one hour; in the muscles, after an obstruction of about three hours, and in the skin, after a still longer time. Death of tissue begins in the form of a coagulation necrosis or of a liquefaction. At the margin of the infarcted area there is a condition of peristasis, with dilated vessels, transudation of fluid and hemorrhages.

FURTHER RESULTS OF CIRCULATORY DISTURBANCES

With the removal of the interference of the local circulation, the fluid in the tissues returns to the blood stream, and small hemorrhages are gradually absorbed. When the circulatory disturbance persists, the tissues remain in a chronic state of subnutrition and of deficient oxygen supply, and secondary changes occur. Changes occur also in the extravasated blood, in the infarcted area and in the vascular supply as a consequence of a permanent obstruction of the vessel. These changes are as follows:

1. Changes resulting from a chronically deficient oxygen supply
   (*a*) Visible and stainable fat and lipoid in the tissues
   (*b*) Hyaline deposits

The appearance of such changes where they are ordinarily not visible is always an expression of a chronically deficient oxygen supply.

2. Changes in the extravasated blood
   (*a*) Decomposition of the red corpuscles, with the formation of hematoidin and hemosiderin and their removal by phagocytes, which are also active in engulfing and removing fat, lipoids and hyalin
   (*b*) Organization of a large hemorrhage by the growth of fibroblasts and vessels into it until finally a scar is formed
   (*c*) Formation of a connective tissue capsule around a hemorrhage too large to be completely organized, the blood within the capsule gradually liquefying until a cyst is formed
3. Changes in the infarcted area

The infarct undergoes organization and is gradually converted into a scar. In some organs, such as the brain, the infarct undergoes liquefaction and an area of softening results.

4. Permanent obstruction of a vessel, with the development of a collateral circulation to circumvent the obstruction
   (*a*) By dilatation of anastomotic vessels already present
   (*b*) By dilatation and growth of capillaries and small vessels until they are of sufficient size to maintain the circulation
   (*c*) By formation of new connecting vessels

As an effect of local circulatory disturbances, whether vasomotor or mechanical or a combination of both, the following changes occur in summary:

1. States of peristasis, prestasis and stasis with transudation of fluid and with hemorrhage
2. Infarction, with the conversion of the infarct into scar tissue or into an area of softening
3. The organization or encapsulation of hemorrhage when this cannot be absorbed
4. The formation of a collateral circulation
5. The deposit of fat, lipoid and hyaline material when the oxygen supply to the tissues is persistently inadequate

CIRCULATORY DISTURBANCES IN THE RETINA

The retina constitutes an organ within an organ, namely, the eye. Its circulation is considerably independent of the general circulation and is regulated according to the needs of the retina by the vegetative system, independent of how the circulation is maintained in other parts of the body. Disturbances in the circulation of the retina are caused here as elsewhere by vasomotor changes, by mechanical means or by both. There is an advantage in studying circulatory disturbances in the retina over studying them in other organs in that changes in the fundus of the eye can be directly seen with a magnification of sixteen times. Although the retina itself is transparent, the vascular tree as well as any deformation of the vessels can be seen in detail; and an opaque substance, such as a transudate, a hemorrhage or a hyaline or lipoid deposit, interferes with the transparency of the retina and is readily visible. In view of the preliminary discussion on circulatory disturbances, if one is investigating such disturbances in the retina the following must be looked for: (1) changes in the walls of the vessels, (2) transudation (edema), (3) hemorrhages, (4) fat and lipoid deposits, (5) hyaline deposits and (6) proliferated glial and connective tissue.

The diseases which produce the vasomotor or mechanical changes responsible for the peristasis, prestasis, stasis, obstruction anemia, infarction and their consequences are manifold and include (1) pathologic changes in the walls of the vessels; (2) functional constriction of the walls of the vessels, either localized or as a part of a generalized arterial constriction, and (3) functional constriction superimposed on anatomic changes in the walls of the vessels or preceding these changes.

A few words are necessary regarding the phagocytic elements which engulf particles of hyalin, lipoid and red blood corpuscles. Their origin is not completely determined. Many phagocytes are undoubtedly histiocytes from the adventitia of the vessels. Some are perhaps glia cells. Other phagocytes have definitely been shown to be derived from the

pigment epithelium layer. The cells of this layer are probably not phagocytic, but in the presence of adventitious substances in the retina they are shed and a proliferation of new cells occurs. The newly formed cells contain less and less pigment; they have ameboid movements and are phagocytic. The still pigmented cells gradually lose their pigment, which then becomes an object of phagocytosis and is carried by the phagocytes to the perivascular spaces.

After this preliminary review on local circulatory disturbances, I shall discuss the two most important pathologic conditions which lead to such disturbances and their consequences, namely, arteriosclerosis and essential hypertension.

ARTERIOSCLEROSIS

It is unfortunate that a number of other terms have been used to describe this disease, such as atheroma, atherosclerosis and arteriolosclerosis, which may lead one to assume that each one stands for a different condition.

Arteriosclerosis, "hardening of the arteries," is one and the same disease, whether the intima presents an ulcer with mush in it, an "atheroma," a localized fibrous thickening or a hyaline thickening of the arterioles. As a definition of arteriosclerosis, I may best use the one given by Aschoff: [5]

> We understand by arteriosclerosis a chronic disturbance of the vessels which manifests itself by deposits of the most varied kinds in the vascular walls and which becomes irreversible on reaching its climax in vessels impaired by changes attending the process of aging with resulting deformation of the lumen and brittleness of the vascular wall.

This definition means that the changes due to aging of the arteries, which also causes a thickening of the vascular wall, must be separated from the changes which constitute arteriosclerosis proper. Aging, or senile fibrosis, according to Aschoff, begins at the end of the "stationary period" in life, which is the early forties. It manifests itself by an increase in connective and elastic tissue of the intima and in a reduplication of the marginal elastic layer. The media also shows an increase in the connective and elastic tissue. This causes a thickening and a hardening of the walls of the vessel. The elasticity of this newly formed tissue is less complete, and there occurs at the same time as the thickening of the wall of the vessel a stretching of the wall in all directions. This results in a dilatation of the lumen and in a tortuosity of the vessel, a characteristic of all senile fibrous vessels.

The arteriosclerotic process proper consists of the atherosis, hyalinosis, lipoidosis and nodular deformation added in irregular distribution

5. Aschoff, L., in Cowdry, E. V.: Arteriosclerosis, New York, The Macmillan Company, 1933, p. 8.

to the aging process of the vessel. The arteriosclerotic process differs somewhat in the various vessels. In an elastic artery with a well developed intima the process begins with the appearance of fat and lipoids in the cells of the intima and a fine dustlike lipoid deposit in the ground substance (lipoidosis). At the same time there occurs a gelatinous transformation of the ground substance. This area of fatty infiltration increases in size; the center becomes mushy and liquefied and constitutes an atheroma. Meanwhile there is localized growth of elastic and connective tissue between the endothelium lining the lumen and the fatty focus, which prevents the atheromatous mush from breaking into the lumen. When the atheroma does break through, an atheromatous ulcer is formed. In the atheromatous focus fatty acids are set free and attract calcium, which is deposited there. This process of lipoidosis, atheromatous formation, calcinosis and nodular thickening of the intima produces a deformation of the wall of the vessel and constitutes the arteriosclerotic process.

In the muscular arteries of the extremities the process is somewhat different. There the primary process is likely to be a deposit of calcium (calcinosis) in the ground substance of the connective tissue surrounding the muscle fibers. As the deposit of calcium increases, it forms plates, which arrange themselves circularly.

In the arterioles and prearterioles the arteriosclerotic process begins differently. Here the primary process consists of a deposit of hyalin and a hyalinization of the basal membrane underneath the endothelial layer. The hyalinization probably begins as a fibrinoid precipitate, which spreads to the adventitia, melting together ground substance, fibrils and cells. Fats and lipoids are deposited in this hyaline material. Because this process of hyalinosis and lipoidosis is characteristic of the smallest arteries and arterioles, it is spoken of as arteriosclerosis. Fundamentally, it signifies changes following a disturbance in nutrition of the arteriole, just as the primary deposit of fat in the elastic vessel and the primary deposit of calcium in the muscular vessel are the results of a local disturbance in nutrition. The arteriolosclerotic process occurs characteristically in certain organs, especially the spleen, kidneys, pancreas and brain, and is not found in the skeletal muscles.

ARTERIOSCLEROSIS OF THE CENTRAL ARTERY OF THE RETINA AND ITS BRANCHES

NORMAL HISTOLOGIC STRUCTURE

The central artery of the retina, according to Hertel, has a lumen of about 210 microns, narrowing to 170 microns at the lamina cribrosa. In infancy the internal elastic layer lies close to the endothelium. As the arterial branches become narrower, this layer becomes thinner and

can be followed as a layer of longitudinal fibers to arteries of a diameter of 10 microns. According to Friedenwald,[6] the internal elastic lamella disappears entirely beyond the primary branches of the central artery. The media consists mainly of circularly arranged fibers, with here and there some which are arranged obliquely. It also contains some elastic fibers. The adventitia is sharply demarcated from the media and contains elastic fibers in addition to the connective tissue; it merges indistinguishably into the connective tissue surrounding the vessel. As the branches get smaller, the muscle fibers are reduced in number until they can hardly be demonstrated in histologic preparations.

RETINAL ARTERIOSCLEROSIS

Histologic studies of arteriosclerosis of the central artery have been made by Raehlmann,[7] Hertel,[8] Harms,[9] Baumgärtner [10] and Bridgett [11] and others. Baumgärtner's contribution is especially valuable. He found that the development of the artery throughout life corresponds to that of other arteries of the same size. There is a normal physiologic thickening of the intima in the ascending period of life consisting of elastic fibers and interstitial tissue, probably containing muscle fibers. This remains without much change during the stationary period of life, that is, until about the end of the fourth decade. The aging process begins in the central artery, as in other arteries, after 45. This process consists here also in a reduplication in the internal elastic lamella into two or three layers and in an increase of connective tissue. Such a senile thickening in the absence of the arteriosclerotic process was found by Hertel, but Baumgärtner considered its occurrence without regressive changes, such as fatty degeneration, rather rare. Bridgett in a study of the central artery of the retina at 200 autopsies found that during the third and fourth decades it was usually but not always possible

6. Friedenwald, J. V., in Cowdry, E. V.: Arteriosclerosis, New York, The Macmillan Company, 1933, p. 363.

7. Raehlmann, E.: Ueber ophthalmoskopisch sichtbare Erkrankung der Netzhautgefässe bei allgemeiner Arteriosklerose, mit besonderer Berücksichtigung der Sklerose der Hirngefässe, Ztschr. f. klin. Med. **16**:606, 1889.

8. Hertel, E.: Beitrag zur Kenntnis der Angiosklerose der Centralgefässe des Auges, Arch. f. Ophth. **52**:191, 1901.

9. Harms, C.: Anatomische Untersuchungen über Gefässerkrankungen im Gebiete der Arteria und Vena centralis retinae und ihre Folgen für die Circulation mit besonderer Berücksichtigung des sogenannten hämorrhagischen Infarktes der Netzhaut, Arch. f. Ophth. **61**:1, 1905.

10. Baumgärtner, H.: Ueber die regressiven Veränderungen der Arteria centralis retinae bei Arteriosklerose, Munich, Thesis, Wiesbaden, J. F. Bergmann, 1914.

11. Bridgett, C. R.: Sclerosis of Central Artery of Retina, Am. J. Ophth. **9**: 725, 1926.

to find a thin, almost homogeneous subendothelial band as well as separation of the internal elastic lamella into a double, and more rarely, into a triple layer. He also found a slight increase in the elastic tissue in the adventitia. At 43 autopsies he found the development of the subendothelial tissue somewhat excessive.

The aging process seems thus to develop in the central artery of the retina in the same manner as it does in other arteries; the arteriosclerotic process is added to this aging fibrosis. Occasionally the arteriosclerotic process is found to occur in young persons. Baumgärtner had a patient of 23 in whom at autopsy fat deposits were found in the intima and one of 33 in whom there was calcification of the elastic fibers in the intima and the media. The arteriosclerotic process begins here also with the appearance of fat and lipoids in the intima. This is followed by formation of atheromas, rupture of the elastic fibers and reparative nodular proliferation of the subendothelial connective tissue; calcium is deposited in the atheromatous areas. Nodular thickening of the wall of the vessel and deformation of the vessel are the results. Baumgärtner found calcification of the elastic fibers in the media with an increase in connective tissue and occasionally also fat and lipoid deposits. The adventitia is affected only slightly or not at all. Hertel, Harms and Baumgärtner found a predilection for the arteriosclerotic process in the region of the lamina cribrosa and in the region of the entrance of the ophthalmic artery into the optic nerve.

The changes in the ophthalmoscopically visible branches of the central artery are similar to those of the main artery but are less common. Friedenwald [12] pointed out "that the ophthalmoscopically visible lesions are merely fringes of a lesion the major portion of which is obscured from view." In the smallest arteries and arterioles, which have no elastic fibers and are not visible ophthalmoscopically, the aforementioned arteriosclerotic process is not found. Here there are found hyalinization and lipoid deposits such as occur in the arterioles and prearterioles of organs like the spleen, kidneys, pancreas and brain. This "arteriolosclerosis" of the retinal arteries occurs in combination with arteriosclerosis of the central artery and its branches under certain conditions, which will be discussed later. It is not, however, part of the picture of arteriosclerosis of the central artery and its branches. In the spleen such an arteriosclerosis is quite common after the age of 10 years and is probably due to local arterial constrictions. It occurs to a marked degree in the kidneys in cases of essential hypertension with renal insufficiency or malignant renal sclerosis. In the retina the hyaline and lipoid deposits in the arterioles and prearterioles occur in association with arterioconstrictive or arteriospastic retinitis.

12. Friedenwald,[6] p. 370.

OPHTHALMOSCOPIC APPEARANCE OF RETINAL ARTERIOSCLEROSIS

In examining the retinal vessels one must keep in mind that the aging process is the same as in other vessels of similar size. It manifests itself in dilatation and tortuosity of the vessels; to these changes is added the irregularly distributed arteriosclerotic process. One must also keep in mind that when looking at a normal vessel with the ophthalmoscope only the blood column is seen and not the wall of the vessel. When there are irregular and patchy changes in the wall they produce an irregularity in the appearance of the blood column. There are, therefore, as a result of the aging plus the arteriosclerotic process, the following signs:

1. A tortuosity of the wall of the vessel. This tortuosity differs from that seen in hypermetropic eyes of young persons and from the congenital variety in that it is more or less angular and does not have the sinuous quality found in the other conditions. In cases of advanced retinal arteriosclerosis it causes the small vessels in the macula to have a corkscrew appearance.

2. Localized variations in the caliber of the vessels. This change is the direct visible expression of localized arteriosclerotic patches. There are all kinds of constrictions in caliber, single or multiple, extending over variable distances, with normal appearing stretches in between the constrictions or with dilatation between them.

3. Localized arteriovenous constriction. This sign was first observed by Gunn.[13] It is seen as an indentation or compression of the vein where it is crossed by an artery. There has been a good deal of discussion as to how the apparent venous constriction is produced and what it signifies. It has been assumed by some authors to be a sign of increase in the general arterial pressure. There is no evidence for this assumption. The most recent contribution is that by Sallmann.[14] He studied the histologic structure of the arteriovenous crossing in a number of persons of all ages with and without hypertension. He found that the artery and vein lie close together where they cross and frequently have a common adventitial coat at this point, an observation previously made by Koyanagi and by Friedenwald. Sallmann found that the vein dips down deep into the retina where it is crossed by an artery, in young persons more so than in older ones. In old persons with or without hypertension there is a thickening of the wall of the vessel and of the common adventitial coat. As the vein dips down, it is hidden by the thickened

13. Gunn, R. M.: Ophthalmoscopic Evidence of (1) Arterial Changes Associated with Chronic Renal Disease, and (2) Increased Arterial Tension, Tr. Ophth. Soc. U. Kingdom **12:**124, 1891-1892.

14. Sallmann, L.: Zur Anatomie der Gefässkreuzungen am Augenhintergrund, Arch. f. Ophth. **137:**619, 1937.

or sclerosed nontransparent wall. Gunn's sign is thus a sign of thickened or sclerosed vessels.

4. Widening of the arterial light reflex. H. Friedenwald [15] pointed out that the width of the light reflex is closely related to the width of the blood column. It would, therefore, be wider in the dilated arteriosclerotic vessel.

These signs, angular tortuosity of the vessels, variations in caliber, apparent arteriovenous constriction and widening of the arterial light reflex, constitute the visible evidence of arteriosclerosis of the retinal vessels. They are found in all degrees of variation. In evaluating them it must be kept in mind that while arteriosclerosis occasionally occurs in young persons, below 40 years of age, it is commonly seen as an addition to the aging process. As the latter begins to manifest itself toward the end of the fifth decade, and as it as well as the arteriosclerotic process are of slow development, it is obvious that the signs of arteriosclerosis described here will become noticeable only in advanced age, in the sixth decade and beyond. This is the normal occurrence. When these signs are found they give visible expression to the degree of the arteriosclerotic process in the retinal vessels themselves; since the central artery of the retina is a part of the cerebral circulation, the signs are a valuable guide to the approximate degree of arteriosclerosis in the brain, and the degree of arteriosclerosis in the retina gives some indication to the degree of arteriosclerosis in the body generally. This is especially true when these signs occur in comparatively young persons, that is, in the fifties or forties or even in younger persons. The wear and tear which blood vessels can withstand are principally dependent on their inherited characteristics, and the signs of wear and tear usually manifest themselves in the senium. When signs of a considerable degree of arteriosclerosis of the retinal vessels are encountered before the senium and in young persons, two etiologic factors must be considered as responsible: first, an inherited deficiency in the wall of the vessel, causing it to give way sooner to the ordinary wear and tear; and, second, an intensification of the wear and tear, causing the vessels to give way prematurely. The one important factor which increases the intensity of the wear and tear of the arterial wall is a persistent increase in blood pressure, especially in the chronic form known as essential arterial hypertension. The finding of signs of arteriosclerosis in the retinal vessels of middle-aged and of young persons calls, therefore, for an immediate investigation as to the presence of a persistent arterial hypertension. Here it is important to make clear distinctions. The signs discussed here, angular tortuosity

15. Friedenwald, H.: The Doyne Memorial Lecture: Pathological Changes in the Retinal Blood-Vessels in Arteriosclerosis and Hypertension, Tr. Ophth. Soc. U. Kingdom **50**:452, 1930.

of vessels, variations in caliber, apparent arteriovenous constriction and widening of the arterial light reflex, are not signs of a general arterial hypertension. They are signs of arteriosclerosis of the retinal vessels. Only their appearance in middle-aged or in young persons serves notice to the observer that something is responsible for their premature occurrence, and the presence of a persistent hypertension may be surmised and must be sought for.

CIRCULATORY DISTURBANCES AS A RESULT OF ARTERIOSCLEROSIS OF THE RETINAL VESSELS

The signs which signify the presence of arteriosclerosis of the retinal vessels do not signify the presence of circulatory disturbances there. Something must be added to produce such disturbances and to create the conditions of peristasis, prestasis, stasis, ischemia and infarction. This something is an interference with the blood flow either in the central artery and its branches or in the central vein and its branches. Such an interference is, as I have previously stated, either mechanical or functional or vasomotor or both. In the central artery and its branches obstruction of the lumen as a result of arteriosclerosis occurs as follows:

1. The local arteriosclerotic lesion extends farther and farther into the lumen until it is completely obstructed.

2. The arteriosclerotic lesion extends into the lumen for a considerable distance, and at its site a thrombus is formed which completely obstructs the lumen.

3. To the arteriosclerotic plaque extending into the lumen there is added a local vasomotor constriction of the vessel, causing complete obstruction.

In the central vein and its branches obstruction of the lumen occurs as a result of phlebosclerosis as follows:

1. The sclerotic process gradually encroaches more and more on the lumen of the vessel, completely obliterating it.

2. To the sclerotic process extending for a variable distance into the lumen there is added a thrombus, which completely occludes the lumen. Obstruction in either the artery or the vein may be complete or incomplete, allowing for some flow of blood. In the artery the obstruction is hardly ever so complete as to cause a total collapse of the vessel; usually a thin plasma stream is still present.

OBSTRUCTION OF THE CENTRAL ARTERY AND ITS BRANCHES

When the clinical picture of obstruction of the central artery was discovered the obstruction was assumed to be due to an embolus, especially as it was frequently found in connection with cardiac disease.

Objections were soon raised to such a view, and the controversy lasted for a long time and is perhaps not over yet. Harms [9] in 1905, in an elaborate study of the reported cases of occlusion of the central artery, came to the conclusion that the presence of a true embolism of the central artery has not been proved anatomically. Still later, in 1925, Scheerer [16] in a critical review came to the conclusion that the anatomic reports of cases of embolism of the central artery in the German literature since 1900 were not able to withstand criticism. He showed that postmortem coagula are frequently found in the central artery of the retina and undoubtedly have been mistaken for emboli or thrombi.

It is necessary to realize that before the early part of this century the differences between bacterial and rheumatic endocarditis were not so well understood. It is now known that emboli do not occur in uncomplicated rheumatic endocarditis. Since occlusion of the central artery occasionally occurs in rheumatic valvular disease, especially in mitral stenosis, such an occlusion cannot be caused by an embolus. More logical is the assumption that such an occlusion in mitral stenosis is caused by a functional constriction of the artery. In cases of acute and subacute bacterial endocarditis in which emboli are continually thrown into the blood stream it is probable that they are occasionally lodged in the central artery or its branches. However, from the literature on bacterial endocarditis it is difficult to determine how often such emboli occur in the central artery in the course of this disease.

CIRCULATORY DISTURBANCES AS A RESULT OF OCCLUSION OF THE CENTRAL ARTERY OR ITS BRANCHES

The clinical picture of occlusion of the central artery is well known. The essential features are: (1) sudden loss of vision; (2) narrowing of the arteries, in which a thin line of slowly moving blood is seen, sometimes having a granular or broken-up appearance; (3) inability to produce arterial pulsation by pressure on the globe, and (4) milky white appearance of the central part of the retina in which the nonedematous fovea appears as a red spot. A small hemorrhage may be found here and there in the retina but is, strictly speaking, not part of the clinical picture.

When the edema has subsided there remains finally (1) a pale atrophic optic disk with sharp distinct edges and (2) very narrow arteries, which are accompanied here and there near the disk by fine white lines for a short distance.

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16. Scheerer, R.: Ueber Vorkommen und Bedeutung freier Blutpfröpfe im Stamme der Zentralgefässe, Arch. f. Ophth. **115**:370, 1925.

The occlusion of a branch of a central artery is characterized by similar symptoms in the area of the retina supplied by that particular branch.

With an understanding of the processes involved in local circulatory disturbances, it is easy to follow the happenings in the retina in occlusion of the central artery, whether it is of mechanical or of functional origin. After the occlusion, there is a reflex constriction of all the arterial branches and, to a lesser degree, of the veins. The arterial constriction immediately creates the conditions for a state of peristasis and prestasis. The terminal units of precapillary arterioles, capillaries and postcapillary venules dilate, and plasma transudes into the surrounding retina. The transuded fluid causes a swelling of the inner layers of the retina. Where the inner layers are missing, in the fovea, the normal color contrasts sharply with the white of the edematous retina. The absence of hemorrhage or its occasional occurrence only is to be explained by the red corpuscles being prevented from entering the terminal units by the obstruction above. When the interruption of the blood supply is maintained for a certain period (said to be not more than half an hour, although this is doubtful), there is an anemic infarction of the inner layers of the retina. The nerve fibers, the ganglion cells and the inner nuclear layer die, and phagocytes make their appearance. Within a period of time, weeks or months, the edema recedes, hyaline and fat deposits appear here and there, any hemorrhage is absorbed and there is an increase in glial and connective tissue which gives the retina a shagreened appearance. Glial and connective tissue also appear as white lines along some of the vessels for variable distances. The end result is a retina with a thin atrophic inner layer in which there are few nerve fibers, few nerve cells and only the single layer of nuclei belonging to Müller's fibers.

The occlusion of any branch of the central artery has a similar train of processes in its wake: constriction of the artery peripheral to the obstruction, dilatation of the terminal units, transudation of plasma and perhaps a few hemorrhages; then follows disappearance of the transudate, hyaline and lipoid deposits, appearance of phagocytes, increase in glial and connective tissue and, finally, atrophy of the part of the retina supplied by that particular branch.

Obstruction of the Central Vein.—Obstruction of the central vein occurs most frequently at the lamina cribrosa, and obstruction of a venous branch, at a point where it is crossed by an artery. It is characterized clinically and ophthalmoscopically by (1) diminution of vision to a variable degree, from the counting of fingers to perception of hand movements in some cases; (2) redness of the optic disk and blurring of its margins; (3) narrowing of the arteries; (4) dilatation and tortuosity of the venous branches, which may be two or three times their

normal caliber; (5) absence of arterial pulsation and of collapse of the veins on pressure on the globe; (6) single and confluent hemorrhages of all sizes and shapes (flame shaped, radial and round), extending widely from the optic disk to the periphery and occasionally into the vitreous, and, later, (7) white or yellowish white deposits of hyalin and lipoids within the areas of hemorrhage.

Obstruction of a branch of a central vein produces a similar train of symptoms localized to the area of the retina which the venous branch drains.

When the obstruction is incomplete, all the signs are less marked.

The circulatory disturbances can be traced here. When the hindrance in the central vein is sufficiently large to obstruct the flow of blood, there is a slowing of the blood stream and a dilatation of the veins above the obstruction. This slowing extends backward to the small venules, capillaries and arterioles. With the slowing of the circulation, the stage of peristasis passes quickly into one of prestasis and then into one of stasis. At the same time there is a reflex constriction of the branches of the central artery. The numerous hemorrhages occur during the prestatic state. When the blood begins to be absorbed, the deficient oxygen supply is responsible for the appearance of hyaline and lipoid deposits. Some of the white spots are found to consist of ganglioform enlargement of nerve fibers. With an incomplete obstruction of the central vein, the same sequence of events occurs.

The further consequences of obstruction of the central vein are: (1) the development of a collateral circulation and (2) atrophy of the inner layers of the retina and hypertrophy of the supporting glial and connective tissue.

The development of a collateral circulation occurs in the manner previously described. The new anastomotic vessels have various forms, straight or curved, connecting the obstructed branch with a neighboring vein, or they form tortuous glomerular networks of all shapes and sizes. Frequently they are found in proliferated connective tissue which projects into the vitreous.

Of great clinical importance is a complication of occlusion of the central vein, namely, secondary glaucoma; but this is not a subject for discussion here.

Obstruction of Very Small Arteries and Veins.—It is necessary to discuss separately the obstruction of small arteries and veins. Such a small vessel may become obstructed either by a local proliferative change in its walls or by functional constriction. When such a small artery is obstructed, there is a localized area of edema, seen as a fluffy white spot; the obstructed vessel may not be visible. It is not an uncommon occurrence and causes a small relative or absolute scotoma. When a small vein is obstructed, one or more hemorrhages are the result. In the

periphery of the retina the blood supply is scanty, and with the aging process the number of capillaries there is gradually reduced. This leads to a thinning of the already thin retina at this point and to the formation of cystoid spaces. Vogt [17] has pictured obliterated vessels leading to peripheral areas of cystoid degeneration.

There is one area in the retina in which retinal capillaries do not exist, namely, the fovea. Obliteration or occlusion of small arterioles leading toward the macula will result in peristatic edema and later in atrophy and cystoid degeneration of the retina at this point and finally in the formation of a hole. This process can be followed occasionally from its inception to the formation of a hole.

A similar process occurs with the occlusion of a small vein in the macula, leading to prestasis with hemorrhages and then to stasis. In the macular area such a hemorrhage after absorption is followed by atrophy of the retina, cystoid degeneration and the formation of a hole. I have been able to follow 2 such cases from the obstruction of a small vein in the macular region to the formation of a hole there.

ESSENTIAL ARTERIAL HYPERTENSION

In discussing arteriosclerosis, I have said that normally the arteriosclerotic process which adds itself to the aging process becomes manifest in old age, in the sixties and seventies, but rarely in the fifties. When arteriosclerosis becomes visible in persons of middle age or in younger persons, one of two factors or both can be held responsible for its early appearance: (1) an inherited deficiency in the walls of the vessels, which causes them to undergo earlier the changes due to wear and tear; and (2) an undue intensification of the wear and tear of the walls of the vessels. As a cause of such an intensification, I mentioned persistent arterial hypertension.

Persistent arterial hypertension occurs in two forms: as an accompaniment of diffuse glomerulonephritis and of malignant renal sclerosis and as essential arterial hypertension. There are distinct differences between the two forms, even as to the appearance of the patient. Persons with diffuse nephritis look pale. Volhard called them "pale hypertensives," in contrast to the full-blooded appearance of persons with essential hypertension, whom he characterized as "red hypertensives." There is also a fundamental difference between the two forms which affects their pathogenesis, course and outcome. This difference consists in the presence of a generalized arterial constriction of the small arteries in diffuse glomerulonephritis and malignant renal

17. Vogt, A.: Ueber zystoide Retinaldegeneration und die begleitenden Liniennetze, und über die optischen Bedingungen der Sichtbarkeit der Zystem, Klin. Monatsbl. f. Augenh. **92**:743, 1934.

sclerosis and in the absence of such a constriction in essential arterial hypertension. I shall discuss essential hypertension and review briefly my conception of this disease.

THE MAINTENANCE OF NORMAL BLOOD PRESSURE

The normal blood pressure of an adult ranges from 110 to 140 mm. of mercury systolic and from 60 to 90 mm. diastolic. An increase above 150 mm. systolic and above 100 mm. diastolic is abnormal. The maintenance of the normal arterial pressure is one of the physiologic norms which has been evolved phylogenetically as an optimum for the organism and which the organism maintains at a constant value within normal limits of variation by means of a regulatory mechanism. This mechanism consists of: (1) effector organs, which are the muscle fibers in the arterial walls in which the contractile tonus is maintained by a balance of certain electrolytes; (2) a vasomotor center in the brain, which regulates the degree of contraction of the muscle fibers; (3) vasomotor fibers, through which the center exerts its regulation, and (4) hormones, especially of the posterior lobe of the hypophysis, another means by which the center in the brain exerts its regulation on the muscle fibers. The whole constitutes a unit mechanism which adapts itself easily to the demands of the body and maintains the arterial pressure at a normal level. This mechanism is ordinarily effective throughout life but is likely to lose its stability in old age when variations in blood pressure become noticeable.

PATHOGENESIS OF ESSENTIAL ARTERIAL HYPERTENSION

In following the course of the blood pressure in a person in whom essential hypertension eventually develops the following sequence of events is observed: In middle life, that is, in the forties or earlier, and in an occasional young person, there are noticed a greater lability and a loss of stability of the normal arterial pressure. There are periods of variation in which the blood pressure rises to 160 mm. and higher, only to return to normal at other periods. The rise in pressure may occur during part of a single day or may last for many days. Essentially these variations are an exaggeration of those occurring in normal persons of that age. With the passing of an indefinite period of time, perhaps a number of years, the variability of the blood pressure still persists, but the pressure returns much less frequently to normal and persists more and more at a higher level, perhaps around 160 or 170 mm. of mercury. With the passing of a further period, the greater variability is still present or is even more marked, but now the blood pressure is permanently high, perhaps around 200

mm. of mercury or higher. The intensification of the wear and tear as a result of the persistently high blood pressure has exercised its influence on the arteries, and both the aging and the arteriosclerotic processes have become intensified, and pathologic changes in the arteries are noticeable. It is evident that the mechanism for the maintenance of the normal arterial pressure is fundamentally defective in such a person and loses its stability at an early age. The cause of the loss of this stability has not been found in any external factor or in any general disease. The only factor known is that of heredity. This factor in essential hypertension has been studied especially by Weitz,[18] and the subject is thoroughly reviewed by Williams.[19]

I conceive essential arterial hypertension to belong to a group of diseases which are characterized primarily by the comparatively early loss of stability of the mechanism for the maintenance of a normal physiologic value. In a previous publication[20] I have included in this group besides essential hypertension also diabetes and chronic simple glaucoma. I define essential arterial hypertension as a disease in which there occurs in some persons in middle life and occasionally earlier a loss of stability in the mechanism for the maintenance of the normal arterial pressure. This loss of stability is the result of an inherited defect already present in the germ plasm. It manifests itself at first by variations in the height of the blood pressure which are exaggerations of the normal variations. Eventually there is a complete loss of ability to maintain the normal blood pressure, which remains now at a high level. With the persistence of the high level of blood pressure, the aging and the arteriosclerotic processes are hastened and appear earlier in life.

THE VASCULAR CHANGES IN ESSENTIAL HYPERTENSION

Since essential hypertension is a disease which begins with a variability in the height of the blood pressure in middle life and extends into old age, it is obvious that any secondary arterial changes will vary with the duration and the degree of the hypertension. In the early years there are practically no changes in the arterial system beyond the ones appropriate for the age period. With the passing of years, the aging and arteriosclerotic processes appear more and more. It is characteristic for this disease that these processes particu-

18. Weitz, W.: Zur Aetiologie der genuinen oder vaskulären Hypertension. Ztschr. f. klin. Med. **96**:151, 1923; Studien an eineiigen Zwillingen, ibid. **101**: 115, 1925.

19. Williams, G. D., in Cowdry, E. V.: Arteriosclerosis, New York, The Macmillan Company, 1933, p. 537.

20. Elwyn, H.: Pathogenesis of Chronic Simple Glaucoma: New Concept of Maintenance of Normal Intraocular Pressure, Arch. Ophth. **19**:986 (June) 1938.

larly affect the vessels of the kidney. In some persons these vessels are exclusively affected; in others, they are affected long before other vessels. It is for this reason that essential hypertension is also spoken of as benign renal sclerosis.

The characteristic changes in the vessels of the kidneys are as follows:

1. In the medium-sized and small arteries there is a thickening of the intima consisting of a multiplication of the elastic lamella with an increase of connective tissue between the lamellae. The muscular layer is reduced in thickness. This process is diffuse throughout the vessels. In the hypertrophied intima there occur regressive changes, namely, hyaline and lipoid deposits in an irregular focal distribution.

2. In the arterioles there are deposits of hyalin and lipoid which cause a thickening of the walls of the vessels. These deposits are not diffuse throughout all the vessels but are found in some and not in others, and in those affected they are not present throughout the whole vessel. These deposits increase in extent with the duration of the hypertension.

Other arteries in the body may be free from any disease. However, with the progress of the hypertension the arteriosclerotic process begins to appear here and there in a focal distribution in other arteries. In many instance the arteriosclerosis is widespread, affecting the aorta and its branches, the arteries of the heart and of the brain. The arteriolosclerotic process is found most frequently in the kidneys only; in some cases and in those in the advanced stage it is found also in the pancreas, less frequently in the liver and occasionally in other organs. The arterioles of the extremities are not affected.

CHANGES IN THE VESSELS OF THE RETINA IN ESSENTIAL HYPERTENSION

In the early stages of essential hypertension, and sometimes even in the advanced stages, there are no changes in the vessels of the retina to indicate a premature aging or the presence of arteriosclerosis. In many cases, perhaps in most, the prolonged intensification of the wear and tear affects the vessels of the cerebral circulation and with it the arteries of the retina. There is then noticed ophthalmoscopically the premature appearance of signs of arteriosclerosis in the retinal vessels, angular tortuosity, variations in caliber, widening of the light reflex and apparent venous constriction where a vein is crossed by an artery. A certain degree of fulness or plasticity of the vessels which makes them stand out clearly is noticed in some cases. There is nothing in these signs which is characteristic of essential hyper-

tension. It is their early appearance and their manifestation to an unusual degree which calls attention to the possible presence of arterial hypertension.

COMPLICATIONS

With its earlier development, arteriosclerosis becomes intensified in years. All the complications mentioned in the discussion on arteriosclerosis of the retinal vessels occur here more frequently and earlier: occlusion of the central artery or its branches and occlusion of the central vein or its branches or a combination of both. Circulatory disturbances leading to peristasis, prestasis, stasis and infarction occur with these complications.

Aside from these more marked complications, there occur occasionally in the retinal vessels in cases of advanced essential hypertension two processes which lead to local circulatory disturbances.

1. A marked degree of tortuosity, dilatation of some tiny vessels and constriction of others are found irregularly distributed in the very small vessels. These irregular dilatations and constrictions lead to circulatory disturbances in small areas and states of peristasis, prestasis and stasis. Small hemorrhages, pinpoint and larger; small patches of transudate, which quickly disappear, and small deposits of hyalin and lipoids, which either disappear or remain, appear in the areas of the retina supplied by these small vessels.

2. The second process is of more serious import. I have said that essential hypertension is a disease in which the mechanism for the maintenance of the normal blood pressure has lost its stability and has become more labile. The loss of stability once begun increases in years. With it there occur occasionally episodes in which without any apparent reason there is a sudden increase in blood pressure, which rises to 250 or 300 mm. of mercury, only to return to the previous level after a variable period of time. During such an episode there may occur temporarily a generalized constriction of small arteries or a localized constriction of the arteries of some individual organ, including also those of the brain and of the eyes. The occurrence of such episodes has long been known. They have been described by Osler[21] as "transient attacks of aphasia and paralysis in states of high blood pressure and arteriosclerosis." These attacks are frequently a precursor of a more serious change in the tempo of progress of the disease.

When these attacks occur suddenly with a generalized constriction of the small arteries and a sudden rise of blood pressure, there is likely to occur a sudden increase in intracranial pressure as a result

21. Osler, W.: Transient Attacks of Aphasia and Paralyses in States of High Blood Pressure and Arteriosclerosis, Canad. M. A. J. 1:919, 1911.

of edema of the brain. This reacts on the eyes as does any increase in the intracranial pressure. Ophthalmoscopically, there is seen a swelling of the disk, which varies from a blurring of its margins to a swelling of several diopters. There is the usual accompaniment of hyperemia of the veins, narrowing of the arteries and hemorrhages. With the subsidence of the attack and the return of the blood pressure to the previous level, the swelling of the disk recedes. The hemorrhages are gradually absorbed and the vessels regain the appearance which they had before the attack. Some hyaline and lipoid deposits may be seen.

Such an attack may be the only one, or it may recur; or, instead of receding, the swelling of the optic disk may remain for months after the acute cerebral symptoms have disappeared and the patient is otherwise quite well.

The occurrence of such an attack of generalized arterial constriction in the course of essential hypertension is significant of a change in the progress of the disease, especially when the occurrence is repeated. The disease is about to lose its benign character and will within a variable period of time pass into malignant renal sclerosis.

THE MALIGNANT PHASE OF ESSENTIAL ARTERIAL HYPERTENSION OR MALIGNANT RENAL SCLEROSIS

In discussing essential hypertension, I mentioned the occasional occurrence in some cases of transient arterial constriction generally or locally in the brain and also in the retina. This transient constriction results in local circulatory disturbances. I come now to the cases of essential hypertension in which there occurs after a long period of hypertension a generalized constriction of the small arteries which is not transient but permanent. In these cases the hypertension occurs earlier in life and is more marked, from 200 to 300 mm. of mercury systolic and from 140 to 190 mm. diastolic. Volhard rendered a great service when he discovered that it is the added general arterial constriction which almost suddenly converts the relatively benign essential hypertension or benign renal sclerosis into the malignant renal sclerosis with progressive renal insufficiency.

Pathologically, the arteriosclerotic and arteriolosclerotic processes are markedly advanced in the kidneys. There are found: (1) a diffuse thickening of the intima and a multiplication of its elastic layers in the larger and smaller arteries; (2) a cellular and fibrous proliferation in the intima of many small arteries forming an obliterating endarteritis; (3) marked hyaline and lipoid deposits in the proliferated intima of the small and smallest vessels, including the arterioles; (4) hypertrophy of the muscular layer in the medium-sized and small arteries; (5) a necrosis of the walls in some of the thickened arteri-

oles, with nuclear disintegration and with evidence of hemorrhage in the walls; and (6) correspondingly severe changes in the glomeruli and tubules.

Pathologic changes in the vessels have been found in other organs, in the pancreas, spleen, liver and brain, but they are much less marked than in the kidneys and are frequently slight.

In the retina the pathologic changes in the vessels have been recently studied by de la Fontaine Vervey,[22] Kyrieleis,[23] Friedenwald[6] and Garsteiger.[24] These investigators have found arteriolosclerosis, that is, thickening of the prearterioles and arterioles due to the deposit in the vessels of hyalin, fat and lipoid. Lipoids were also found in the small vessels of the optic nerve. There was also hypertrophy of the muscular coat. In addition to the arteriolosclerosis, there is an intensification of the aging and arteriosclerotic processes in the larger vessels, with nodular thickening of the intima to varying degrees. With these changes in the vessels there are found all the pathologic changes of arterioconstrictive retinitis: edema, hemorrhage, cystoid spaces filled with plasma and fibrin, hyaline, fat and lipoid deposits, phagocytes and ganglioform swelling of nerve fibers.

Ophthalmoscopically there are seen: (1) signs of advanced arteriosclerosis and arteriolosclerosis, consisting of angular tortuosity of the vessels, especially the small ones, variations in the caliber of the lumen, apparent constriction of veins where they are crossed by arteries and a sharp and bright light reflex with irregularity in its width; (2) arterioconstriction, as shown by the narrowed arteries, especially the smaller branches; (3) signs of local circulatory disturbances of acute character, consisting of edema, hemorrhages, cotton wool patches, blurring of the margins of the optic disk and distinct papilledema, and (4) sharp white spots of hyaline deposits, glistening spots and the star figure in the macular area.

Pathologic changes occur also in the vessels of the choroid, and choroidal arteriosclerosis is frequently seen.

Complications of arteriosclerosis, occlusion of the central artery or vein or of their branches, occur in some cases.

The whole clinical picture in the fundus represents the local circulatory disturbances as a result of a persistent and varying arterial

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22. de la Fontaine Vervey, B. C.: Ueber die Arteriolosklerose der Netzhaut und ihre Bedeutung für die Genese der Retinitis albuminurica, Klin. Monatsbl. f. Augenh. **79**:148, 1927.

23. Kyrieleis, W.: Ueber die Arteriosklerose von Netzhaut, Aderhaut und Sehnerv sowie ihre Bedeutung für die Pathogenese der Retinitis albuminurica, Arch. f. Augenh. **103**:161, 1930.

24. Garsteiger, H.: Ueber histologische Befunde am Auge bei Nieren- und Blutdruckveränderungen, Klin. Monatsbl. f. Augenh. **99**:604, 1937.

constriction which has suddenly been added as a malignant factor in the course of an essential hypertension. There is no doubt at this stage of knowledge that the general arterial constriction is responsible for the changed character of the disease, for the appearance of renal insufficiency and for the cerebral and the retinal symptoms. Klemperer and Otani,[25] in discussing Volhard's angiospastic theory from the pathologist's point of view, remarked:

> It is conceivable that a constitutional or acquired angiospastic factor could be the reason for the severity and especially for the acceleration of the vascular process which is the outstanding characteristic for the differentiation between vascular diseases of the kidney with and without functional insufficiency.

CONCLUSION

I have reviewed two of the most important diseases of the retinal vessels which are responsible for circulatory disturbances in the retina. These diseases have two components: (1) organic, with changes in the vessels which interfere with the normal blood flow, and (2) functional, leading to constriction of the vessels. Whether the interference with the normal blood flow is due to organic changes or to functional constriction, the result is the production of local circulatory disturbances in the retina which are similar. These disturbances follow the dilatation of the terminal units of the vascular system, precapillary arterioles, capillaries and postcapillary venules, with constriction of the artery above. Depending on the degree of constriction and dilatation, there occurs peristasis with transudation of fluid, prestasis with hemorrhages, stasis with stoppage of the circulation or infarction. With the release of the constriction and the resumption of the normal blood flow, the transuded fluid and the hemorrhages are absorbed. When the interference with the blood flow persists but the blood supply to the retina is not completely restricted, the chronic suboxidation and subnutrition of the retina manifests itself by deposits of hyalin, fat and lipoids. When infarction occurs, collateral circulation with the formation of all forms of anastomotic vessels is established whenever possible. Hemorrhages that are too large to be absorbed stimulate connective tissue formation and are either organized and converted into scars or are encapsulated.

These consequences of the dilatation of the terminal vessel units and the constriction of arteries above appear in the retina in various forms in the diseases discussed. They can be seen and studied in the retina better than anywhere else in the body. An appreciation of the manner of their production helps in the understanding of the diseases so far as they affect the retina.

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25. Klemperer, P., and Otani, S.: "Malignant Nephrosclerosis" (Fahr), Arch. Path. **11**:60 (Jan.) 1931.

PRIMARY GLIOMA OF THE OPTIC NERVE

REPORT OF A CASE

CARL W. RAND, M.D.

RODMAN IRVINE, M.D.

AND

DAVID L. REEVES, M.D.

LOS ANGELES

Largely as a result of Hudson's [1] comprehensive paper, tumors of the optic nerve have been divided into those of the nerve itself and those of the nerve sheaths. At present most pathologists consider the first group to be gliomas of the spongioblastic type and the second to be endotheliomas or meningiomas and the various fibromas, with rare instances of sarcoma and angioma. It is with the first group, the primary intraneural tumors of the optic nerve, that we are at present concerned.

Glioma of the optic nerve is rare. Less than 300 examples of this tumor have been described in all the medical literature.[2] During a period of thirty-six years at the Massachusetts Eye and Ear Infirmary only 4 such tumors were removed from 669,857 new patients. From the Wills Hospital in Philadelphia DeLong [3] reported that during a period of twelve years only 1 such tumor was found in 230,742 patients admitted. The ratio between gliomas of the optic nerve and sarcomas of the choroid has been found to be about 1 : 200.[2] Hudson [1] observed that primary glioma of the optic nerve was more frequent in the female sex and occurred in the first decade of life in about 75 per cent of the cases. In 1 instance, however, the tumor was discovered as late as the sixtieth year.

Investigation of the literature reveals a confusion of terminology and emphasizes the not infrequent involvement of the intracranial portions of the nerves and chiasm by the neoplasm.

Embryologically, the optic nerve arises from an invagination of the primitive cerebral vesicle and therefore contains all the adult forms of neuroglia. For this reason the same variety of neuroglial tumors

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From the Department of Surgery of the School of Medicine of the University of Southern California, and the Children's Hospital (Dr. Reeves).

1. Hudson, A. C.: Roy. London Ophth. Hosp. Rep. (pt. 3) **18**:317, 1912.
2. Verhoeff, F. H., in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B. Hoeber, Inc., 1932, vol. 3, p. 1029.
3. DeLong, P.: Am. J. Ophth. **17**:797, 1934.

found in the brain might be anticipated in the optic nerve. Up to the present time, however, the polar spongioblastoma is the type invariably described. Although instances of fairly rapid growth during the course of several months are not rare, glioma of the optic nerve, in contradistinction to the retinal glioma grows slowly in the vast majority of cases.

Glioma of the optic nerve presumably advances by direct extension along the intraneural portion of the nerve, does not invade the dura and does not metastasize. Although extension to the chiasm and deeper structures of the brain may occur fairly often, invasion of the disk has been reported in only 4 or 5 instances. The tumor may spread from the intraorbital to the intracranial portion of the optic nerve through the optic foramen, or from the intracranial to the intraorbital portion of the nerve. In the majority of cases reported the neoplasm has occurred in the intraorbital portion. The growths in 20 per cent of Hudson's cases involved both the intraorbital and the intracranial portion.

One of the earliest reported examples of extension into the chiasm was that of Heymann [4] in 1842, a "neuroma nervi optici." The first reported attempt at intraorbital enucleation for sarcoma of the nerve was made by Ritterich [5] in 1861, with fatal results. At autopsy an enormous enlargement of the chiasm was found. Subsequently such growths were treated surgically, among them 3 described by von Graefe,[6] a "sarcoma myxomatodes" and a "myxoma" in 1864 and a glioma in 1866. The last-mentioned growth occurred in a child of 6 who had exophthalmos due to a tumor of the nerve. At operation the globe was removed and the retrobulbar tumor partially extirpated. Meningitis followed, and autopsy revealed what Virchow termed a "gliosarcoma" involving the intracranial portion of both optic nerves, the optic chiasm and the base of the brain.

In 1922 Dandy [7] stressed the fact that a high proportion of intraorbital tumors of the optic nerve may and do extend into the cranial chamber. He emphasized the futility of operations on the orbital part of such tumors, pointing out that a false sense of security may result and more complete surgical intervention by the intracranial approach be delayed until the condition has become hopeless.

Verhoeff [2] observed that Hudson [1] was unable to discover any reported cases of orbital recurrence, even though in many cases the

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4. Heymann, A.: De neuromate optici, Berlin, 1842.

5. Ritterich, F. P.: Weitere Beiträge zur Vervollkommnung der Augenheilkunst, Leipzig, C. F. Winter, 1861, p. 57.

6. von Graefe, A.: Arch. f. Ophth. **10**:193, 1865; cited by Martin, P., and Cushing, H.: Arch. Ophth. **52**:209, 1923.

7. Dandy, W. E.: Am. J. Ophth. **5**:169, 1922.

removal had been incomplete. Verhoeff also found, on the basis of clinical and postmortem evidence, that in most cases the tumor originated in the intraorbital portion of the nerve but soon extended through the optic canal to involve the chiasm. The lack of orbital recurrence is then unimportant in the face of possible intracranial extension of the

Fig. 1.—Preoperative photograph of the patient taken on April 13, showing the outward and downward exophthalmos of the right eye.

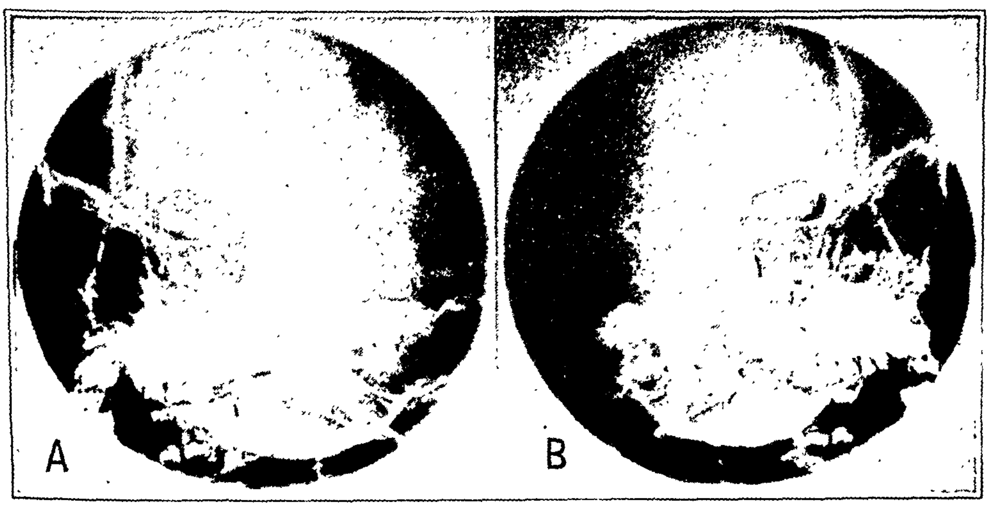

Fig. 2.—Roentgenograms of the optic foramens, taken on April 11: *A*, dilated right optic foramen, and *B*, normal left optic foramen.

neoplasm, which leads to a fatal termination once the chiasm is involved. It is surprising that the early examples of intracranial extension failed to leave much impression on many subsequent writings concerning these tumors.

REPORT OF CASE

History.—J. P., a boy of 4½ years, was admitted to the Children's Hospital from the outpatient department on April 8, 1938, with a history of slowly progressive unilateral exophthalmos, of the right eye, during the preceding year. Coincidentally, there occurred an outward and downward protrusion of the eye, with no apparent distress to the patient. The child was otherwise well. Nothing of significance was discovered in the family or the past history.

Examination.—On admission to the hospital the child appeared well and active except for exophthalmos of the right eye and was little concerned about the con-

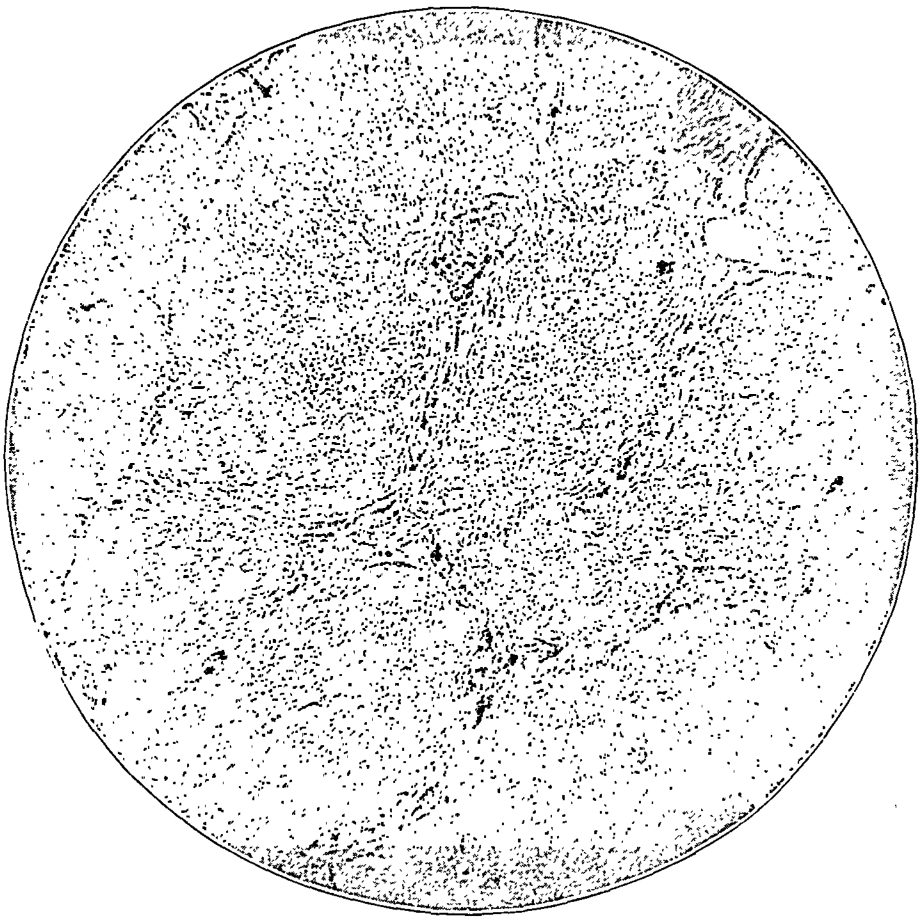

Fig. 3.—Low power photomicrograph of a cross section through the glioma of the optic nerve, showing bands of connective tissue representing the normal fascicular architecture of the nerve with intervals between occupied by typical gliomatous tumor.

dition of the eye. The globe was displaced downward and outward (fig. 1). There was marked limitation of upward movement and slight limitation of inward movement. No resistance was felt when the eye was pushed back into the orbit. The exophthalmos was nonpulsatile. The right pupil was slightly dilated and reacted sluggishly to light. Vision in this eye was reduced to 2/200. Projection of light was good in all fields. The intraocular tension was normal to palpation.

The ophthalmoscope revealed clear media and a slightly grayish optic disk, with definitely fuzzy margins. The retina was somewhat pale on the nasal side of the disk, giving the appearance of having been stretched. The macula had a slightly granular appearance. The arteries were normal; the veins were questionably dilated. There was no pulsation in the vessels. There were no hemorrhages or exudates. The left eye was normal, with vision of 20/20. Both corneal reflexes were equal and active. Satisfactory visual fields could not be obtained because of the inability of the patient to cooperate. The results of general neurologic and physical examinations were otherwise entirely negative.

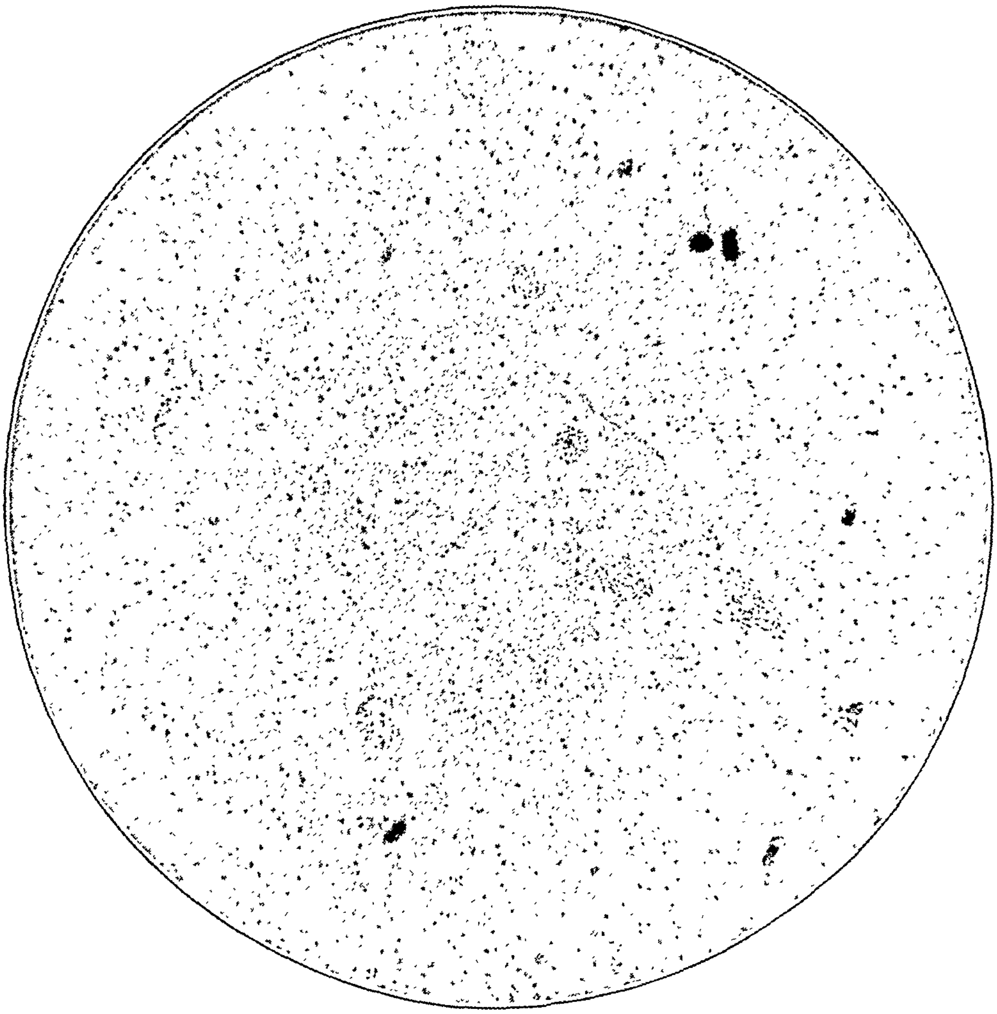

Fig. 4.—Low power photomicrograph of a cross section taken through the normal appearing nerve sectioned near the chiasm.

Roentgenograms of the optic foramens, taken on April 11, as interpreted by Dr. Rolla G. Karshner, revealed a definite enlargement of the right optic canal (fig. 2). Examinations of the urine and blood gave negative results.

First Operation.—In view of the findings, a diagnosis of primary glioma of the right optic nerve in the intraorbital portion with extension toward the chiasm was made. An intracranial exploration was carried out on April 27 by one of us (Dr. Rand), at which time a right frontal craniotomy and section of the nerve about 1 cm. from the chiasm was performed. Both optic nerves were clearly seen. Starting about 1.5 cm. from the chiasm, the right nerve was seen to be symmetri-

cally swollen through the optic foramen. The orbital roof was removed; the nerve was sectioned anterior to the foramen and removed. The osteoplastic flap was then closed in the usual fashion, and the patient was given 200 cc. of whole blood by the multiple syringe method at the conclusion of the procedure. The postoperative course was essentially uneventful.

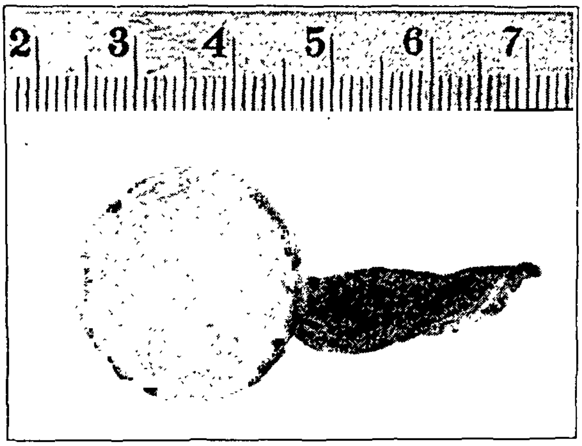

Fig. 5.—Photograph of the eyeball and remaining portion of the optic nerve and tumor attached to the globe, removed on May 24.

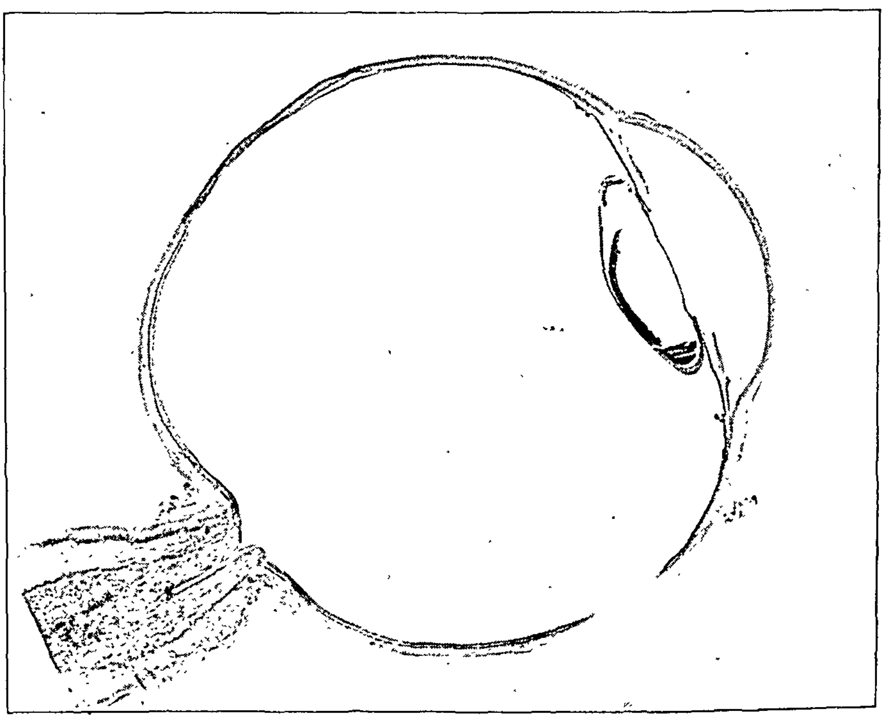

Fig. 6.—Low power photomicrograph of a sagittal section taken through the globe and optic nerve where it enters the globe.

The specimen obtained at the first operative procedure, April 27, consisted of a somewhat fusiform, grayish red enlargement of the optic nerve, measuring 1.3 by 0.8 by 0.5 cm. A solution of formaldehyde was used as a fixative. The orbital

portion was noticeably wider than that removed at a distance of about 1 cm. from the chiasm, where it appeared normal in size. The gliomatous enlargement was smooth and moderately firm, though somewhat elastic in consistency. The dura was not adherent.

Microscopic Examination.—A cross section of the nerve at its widest portion revealed the fascicular pattern of the nerve preserved though greatly accentuated (fig. 3). The pear-shaped spongioblastic cells with their delicate processes were seen to invade not only the nerve fibers but the connective tissue stroma as well. In the region of the septums the blood vessels were often increased and dilated.

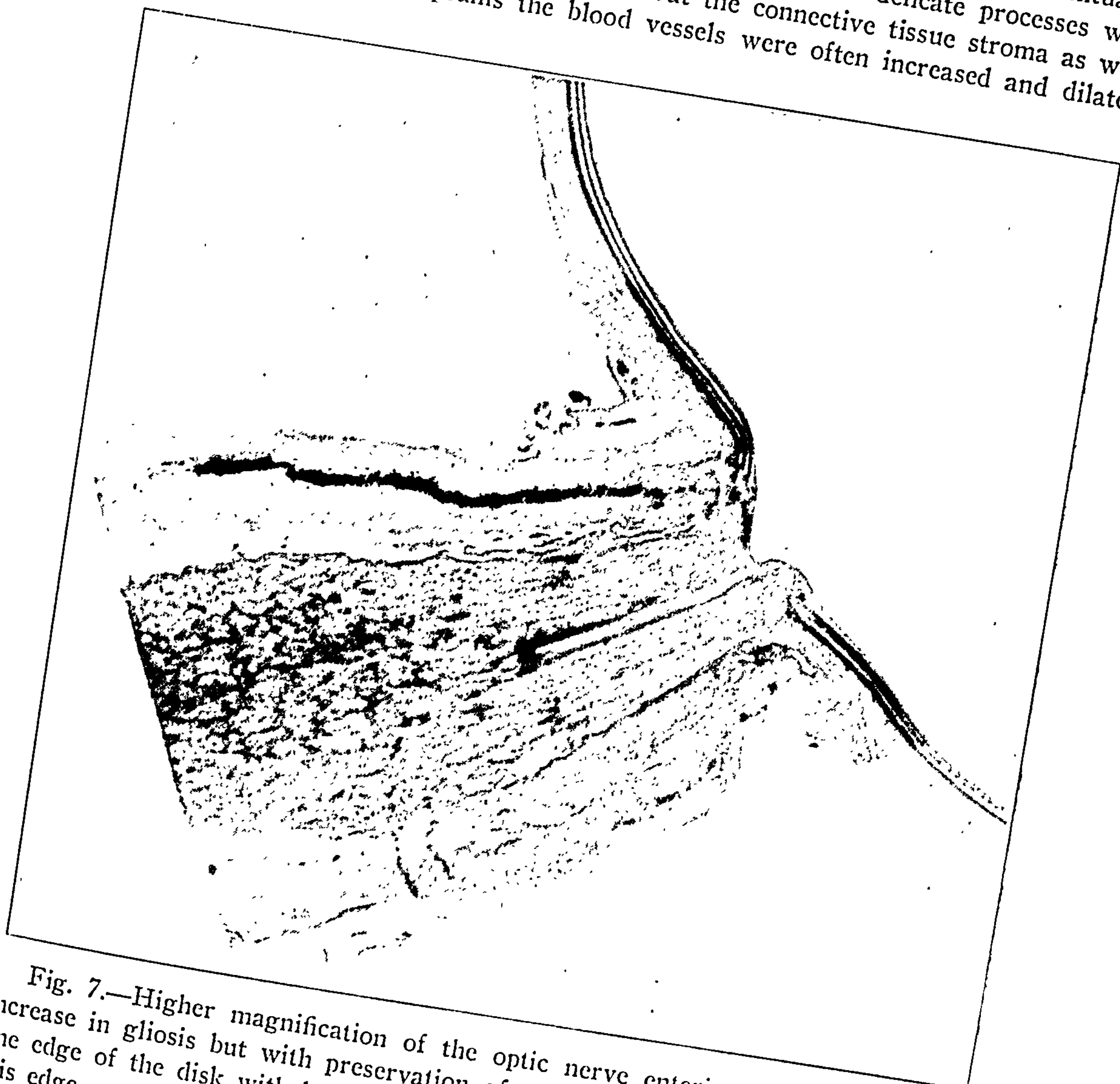

Fig. 7.—Higher magnification of the optic nerve entering the globe showing increase in gliosis but with preservation of normal fasciculi. There is gliosis at one edge of the disk with loss of pigment epithelium for a short distance beyond this edge.

In some places their walls were thickened and hyalinized and gave an angiomatous appearance to the neoplasm. Vacuolation of the cells was evident here and there, leading to the formation of a few small cystic cavities. Some of the cells gave the appearance of being astrocytes, but the polar spongioblastic character of the predominating cell was evident. The only normal nerve tissue remaining was found at the periphery of the section, and this revealed moderate fibrosis of the nerve sheath. A section through the fairly normal appearing narrow por-

tion of the nerve showed a small amount of fibrinous polymorphonuclear exudate at the periphery. There was no evidence of neuroglial proliferation through the nerve fibers or connective tissue stroma (fig. 4). The diagnosis seemed obviously that of primary glioma of the optic nerve, spongioblastic in type.

Second Operation.—Since the greater part of the orbital nerve had not been removed at the first operation and miscroscopic examination had revealed the nature of the tumor, it was decided to remove the globe with the remaining portion of the nerve. This procedure was delayed until healing from the first operation had progressed sufficiently that subsequent enucleation would not be dangerous

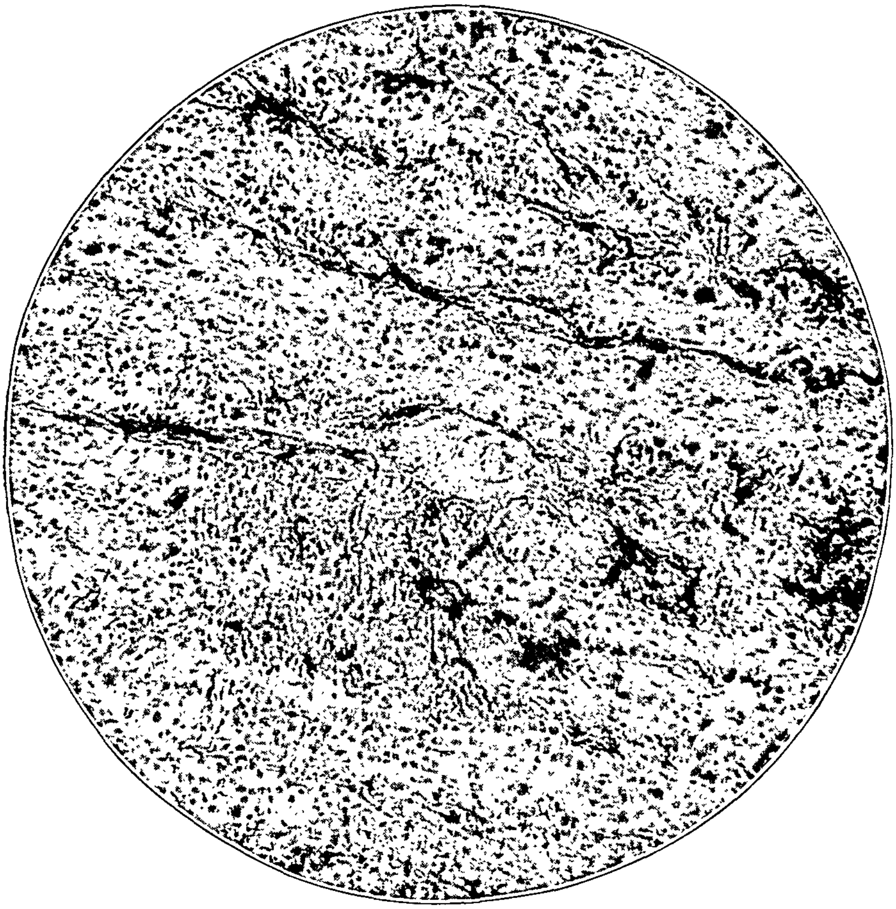

Fig. 8.—High power photomicrograph of detail from figure 7, showing gliosis with preservation of the normal pattern.

from the point of view of infection from the conjunctiva. On May 24 one of us (Dr. Irvine) enucleated the eye with the remaining portion of the nerve attached. The patient made an uneventful recovery and was discharged from the hospital on June 3.

The specimen removed on May 24 consisted of the eyeball and optic nerve (fig. 5). The eyeball appeared normal. The nerve was more or less uniformly dilated, measuring 2.5 cm. in length and 8 mm. in diameter at its widest portion. Except for a sudden constriction to normal size as it entered the sclera, the enlargement of the nerve extended to the globe. Over its entire course the

enlargement was superficially cystic, particularly near the globe. After the globe was fixed in a solution of formaldehyde, it was opened and found to be essentially normal. The retina was in situ, and no gross pathologic process was seen at the disk.

Fig. 9.—Photograph of the patient taken on May 11, fourteen days after intracranial section of the right optic nerve.

Fig. 10.—Photograph of the patient taken on June 1, two days before his discharge from the hospital.

Microscopic Examination.—The pathologic changes were limited to the optic nerve, the eyeball being normal (figs. 6 and 7). The nerve was greatly increased in size. The normal pattern of the nerve was maintained to a great extent, the enlargement being partially due to an increase in neuroglia within the interseptal spaces but for the most part due to marked meningeal reaction.

The outstanding change in the nerve proper was the increase in glial cells (fig. 8). For the most part these varied from round to spindle shape, with a rather clear nucleus containing a nucleolus and rather prominent chromatin. A finely reticulated matrix could be made out, which was more apparent toward the periphery, where there was a suggestion of fine vacuolation in excess of that over the normal neuroglial mantle. Cross section revealed the normal pattern more or less intact and the axis-cylinders present in the periphery. Some of the glial cells were large, approaching a giant cell type.

The leptomeninges were distended by marked proliferative reaction, characterized by a large amount of collagen arranged more or less in parallel thick fibers. Whether or not part of the dense cellular reaction represented proliferation of tumor cells in the vaginal space could not be definitely ascertained but seemed unlikely, as the change in the nerve at this point was rather slight.

The lamina cribrosa curved backward slightly and was questionably thickened as a result of a slight increase in the spindle cells. The optic disk showed some gliosis at one edge, and for a short distance beyond this edge there was a loss of pigment epithelium. The appearance here was somewhat like that of supratraction of the retina due to myopia. A persisting tag of hyaloid artery remained on the disk in some of the sections. The appearance of the disk ophthalmoscopically was undoubtedly the result of the gliosis. The retina appeared normal. The retinal ganglion cells seemed slightly diminished in number.

Slides were sent to Dr. Verhoeff, who confirmed the diagnosis, saying that this was undoubtedly a glioma of the optic nerve such as he has described [2] as the finely reticulated type.

DIAGNOSIS

An intraorbital tumor of the optic nerve can usually be diagnosed with much more certainty than can primary intracranial neoplasm of the nerve. Proptosis and slowly progressive loss of vision are the most common manifestations of an intraorbital tumor. Impairment of vision usually precedes proptosis in cases of glioma, while the reverse is true with endothelioma or fibroma. Because the tumor grows slowly, visual disturbances may begin many years before the onset of proptosis. The direction of displacement of the globe usually coincides with the direction of the orbital axis, but exceptions are noted if the growth becomes large. Limitation of motion of the globe may or may not be present and is more often noted in cases of endothelioma and fibroma than in those of glioma.

Secondary atrophy of the optic nerve is the most common abnormality found on ophthalmoscopic examination. Papilledema may occur with or without evidence of atrophy and is most frequently observed in association with a growth lying close to the globe or in cases in which intracranial pressure is increased.

According to the literature, a normal fundus is occasionally seen in the presence of an intraorbital tumor of the optic nerve. This was noted in 3 of the 118 cases collected by Hudson,[1] the growth in each of the 3 instances being an intraneural glioma. In slightly more than

half of his cases atrophy of the optic nerve was found; the terms optic neuritis and papilledema were used in describing the condition in the remainder.

A roentgenogram of the optic foramen is a most important diagnostic aid in these cases, as the foramen may be considerably enlarged. Without evidence of orbital extension, such as enlargment of the optic foramen and exophthalmos, a diagnosis of primary intracranial glioma of the optic nerve probably can never be made with certainty without surgical exploration. Martin and Cushing [8] were able to diagnose such a tumor preoperatively in only 1 of 7 cases. Atrophy of the optic nerve and slowly progressive loss of vision in one or in both eyes are frequent findings. The visual fields may show bitemporal or homonymous hemianopia, or total blindness in one eye and hemianopia in the other. Roentgenograms may show deformation of the sella turcica and enlargement of one or of both optic foramens. The absence of such evidence, however, is no proof that the lesion may not have extended through the optic foramen, because instances of such extension without deformation of the canal have occurred. Unless the growth becomes large enough to cause an increase in intracranial pressure, papilledema is not produced by a tumor confined to the chiasmal or prechiasmal portion of the optic nerve.

GROSS AND MICROSCOPIC PATHOLOGIC PICTURE

As described by Grinker,[9] the glioma on gross appearance usually has a smooth surface, is somewhat spindle shaped and is covered by easily detached dura. It may, however, appear as a spherical enlargement of the nerve, a solid, irregular thickening along its entire course, or more often as several small nodules separated by thick tubular tissue. Although the dura is rarely penetrated, it may be thinned out and contain tumor tissue on its inner surface. The diameter, of course, is variable, often measuring from 1 to 2.5 cm. The sectioned enlargement discloses either a diffusely thickened optic nerve recognizable as such or an optic nerve peripherally compressed by a grayish red tumor nodule.

Sattler [10] and Verhoeff [2] have shown that when the tumor infiltrates to the papilla it may be visible ophthalmoscopically as a small papillary cyst. Variable-sized cysts filled with mucoid material are not infrequently seen. These cysts, containing a homogeneous substance previously considered mucin, explain the old term myxoma. According

8. Martin, P., and Cushing, H.: Arch. Ophth. **52**:209, 1923.

9. Grinker, R. R.: Tumors of the Optic Nerve, Arch. Ophth. **4**:497 (Oct.) 1930.

10. Sattler, H.: Die bösartigen Geschwülste des Auges, Leipzig, S. Hirzel, 1925.

to Fleischer and Scheerer [11] and Sattler,[10] however, this substance does not stain with mucicarmine or mucihematin. Verhoeff [2] expressed the belief that the cysts which contained serous fluid, small amounts of fibrin and a few red blood cells arise from a fusion of the vacuoles found in the reticulum. Löhlein,[12] on the other hand, was of the opinion that the cysts are the result of areas of degeneration secondary to the marked degenerative and hyaline changes of the walls of the vessels. The cysts may be large. In fact, in Adamuck's [13] case the entire tumor comprised a cyst.

The microscopic appearance of these gliomas created great diversity of opinion among the earlier writers on the subject. They were regarded as mesoblastic, epiblastic, malignant or benign, and again of the nature of a hypertrophy rather than a true new growth. They were referred to by such names as sarcoma, myxosarcoma, glioma, gliomyxosarcoma, adenoma and endothelioma. In 1879 Willemer [14] assembled from the literature the reports of 25 cases in which the most variable histologic structures were described, though many of the growths were doubtless true gliomas of the nerve. In 1892 Sattler [15] expressed the belief that of the essential tumors of the optic nerve, the majority conformed to a type which he called myxofibroma, while only a small portion were endotheliomas.

Hudson [1] in 1912 collected and analyzed all previously reported cases of tumor of the optic nerve. Fortunately, he simplified the classification by dividing the tumors into three groups: those showing gliomatosis, those showing fibromatosis and those of endothelial origin. The second and third groups, of course, consisted of tumors which arose from the nerve sheath or their meningeal envelops, but the first group, tumors exhibiting glial proliferation, consisted of 118 tumors, the larger number of which had been described as myxosarcomas of one form or another. Interestingly enough, in all but 1 of the 11 cases in which there was a postoperative fatality with subsequent autopsy, an extensive intracranial involvement of the nerves and chiasm, often with more or less invasion of the brain, was the invariable postmortem observation. Moreover, it was evident that in 14 other instances symptomatic evidence of intracranial disease had already appeared by the time the cases were reported.

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11. Fleischer, B., and Scheerer, R.: Arch. f. Ophth. **103**:46, 1920.
12. Löhlein, W.: Arch. f. Ophth. **73**:335, 1914.
13. Adamuck, S.: Arch. f. Augenh. **28**:129, 1894.
14. Willemer, F.: Arch. f. Ophth. **25**:161, 1879.
15. Sattler, H.: Ueber die eigentlichen Sehnerventumoren und ihre chirurgische Behandlung, in Beiträge zur Chirurgie: Festschrift gevidmet Theodor Billroth von seinen dankbaren Schülern, Stuttgart, [Hoffmann], 1892, p. 314.

Byers[16] in his publication of 1914 expressed the belief that tumors growing within the dural sheath could not be separated definitely into two divisions. He pointed out that such growths essentially represented an overgrowth of the fibrous connective tissue elements of the optic nerve and that almost every phase of development, from the embryonic myxomatous type to the fine fibrous bands described by the term fibromatosis, could be found. In the second group of neoplasms he attributed the changes to a proliferation of local endothelial cells and classified the growths as endotheliomas. He further added that the two groups were histogenetically related.

At the present time it seems rather generally conceded that these gliomas of the optic nerve are spongioblastic in type. The similarity of these growths caused Verhoeff[2] to state that there could be little doubt but that they were all of the same nature. The same elements could be seen in all of the tumors but in different proportions and arrangement, and all were primarily composed of neuroglia. Verhoeff[2] classified them into three groups, with transitional forms between either one or both of the other groups. The first group he designated as the finely reticulated type with irregularly running fibrils comprising a matrix for small, oval nucleated cells containing a small amount of feebly staining cytoplasm, frequently vacuolated, a type similar to but not identical with the neuroglia of the normal optic nerves. The second group he called the coarsely reticulated type, which appeared to be an exaggeration of the first, with heavier fibrils, larger intracellular vacuoles and a greater tendency to cystic formation. His last group he designated as the spindle cell, or coarsely fibrillated. type, consisting of coarse neuroglia fibrils, many of which were partly spiral in form, with neuroglial cells lying between them.

Although on occasions the vascularity of these gliomas has been sufficient to have them mistaken temporarily for angiomatous lesions, ordinarily the tumor is not very vascular. Numerous thin-walled, dilated vessels are not infrequently described and are usually found in the septums, the common locality for vascular changes in the optic nerve. Hyaline degeneration of the walls of the blood vessels and secondary hemorrhages within them are not uncommonly noted. Although there is great variation of the pia-arachnoid and the extension of its septums into the nerve, they are usually greatly thickened and composed of irregular masses of collagenous tissue into which glia fibers have grown in all directions.

Occasionally a myelin sheath may persist, and small, normal appearing bundles of axis-cylinders appear intact in the peripherally com-

16. Byers, W. G. M.: Tumors of the Optic Nerve, J. A. M. A. **63**:20 (July 4) 1914.

pressed remnant of the optic nerves, but usually the optic nerve fibers are spread apart by the tumor and demyelinated early.

Whether the growth is intraorbital or intracranial, the descriptions of fundamental cell type have been generally similar. The cells are fairly long and spindle shaped, with oval nuclei and processes arising from each end. Sattler [10] described them as oval cells with corkscrew-like processes; Sourdille,[17] as bipolar cells with two prolongations, and Favaloro,[18] as bipolar fusiform cells with short, broad expansions, forming a syncytium.

Verhoeff [2] first described as cytoid bodies rounded or elongated hyaline masses. The largest may attain a cross diameter of 0.025 mm., and they may reach a length of 0.10 mm. None of the bodies contained definite nuclei, although occasionally, after staining with alum hematoxylin, some resemblance to a nucleus was produced by a portion of the substance staining more deeply than the rest. These pseudonuclei never showed nuclear membranes or contained chromatin nets or nucleoli. Continuous from them in a few instances were long processes which could be followed into a spindle-shaped neuroglial cell. Verhoeff was of the opinion that they were atypical or giant neuroglial fibers. Martin and Cushing,[8] on the other hand, considered them merely masses of coalesced glial fibers.

According to Grinker,[9] these tumors contained numerous mitotic figures, but Verhoeff [2] has never found any neuroglial nuclei undergoing either direct or indirect division. He accounted for this on the basis of the slow rate of proliferation.

Favaloro [18] found small round cells without processes and with two or more nuclei, which he considered to be oligodendroglia. He also discovered astrocytes mixed with bipolar fusiform cells. Enriquez [19] and Marchesani [20] reported astrocytes and oligodendroglia in the normal optic nerve, but at the present time no definite differentiation of the spongioblasts into the more advanced glia cells has been reported in tumors of the optic nerve.

As the optic nerve contains all of the adult forms of neuroglia, it is not unreasonable in gliomatous tumors of this nerve to expect characteristics and differences in malignancy similar to those found in cerebral gliomas. The fact that at the present time these tumors have invariably been reported as spongioblastic does not, of course, mean that other gliomatous types will not be found subsequently. It is to be remembered, however, that certain of the gliomas are discovered more

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17. Sourdille, G.: Arch. d'opht. **24**:87, 1904.
18. Favaloro, G.: Ann. di ottal. e clin. ocul. **56**:619, 1928.
19. Enriquez, L.: Bol. Soc. españ. de hist. nat. **26**:1, 1926.
20. Marchesani, O.: Arch. f. Ophth. **117**:575, 1926.

often in some localities of the brain than in others. Thus the polar spongioblastoma predominates in the region of the third ventricle, optic chiasm and optic nerves, while the medulloblastoma is most commonly found in the central cerebellar region of children, and the glioblastoma multiforme, preeminently the glioma of adult life, occurs almost invariably in the cerebral hemispheres. For this reason one could scarcely be surprised if the polar spongioblastoma were the glioma of the optic nerve and chiasm invariably found.

As these gliomas of the optic nerve are seen much less frequently than cerebral gliomas and facilities for the specific staining methods are not always available, it is not surprising that a satisfactory understanding of their classification and variation in malignancy has not been obtained. Even though various spongioblastic tumors may appear similar, differences in rapidity of growth in the group is to be expected, just as in the different groups of cerebral gliomas.

TREATMENT

Complete surgical removal of the involved nerve offers the best prognosis for preservation of life and vision. As emphasized by Dandy,[7] the fact that a high percentage of intraorbital tumors of the optic nerve extend into the cranial chamber increases the surgeon's responsibility in determining whether or not there is intracranial extension before he is content with orbital removal. Unfortunately this often cannot be determined with satisfaction preoperatively. Enlargement of the suspected optic foramen would seem sufficient evidence to justify the assumption that the tumor has invaded the intracranial portion of the nerve, although, as mentioned previously, absence of such enlargement is no proof that this has not occurred. Should local enucleation be done, a section of the cut nerve should be studied to determine if possible whether or not all the tumor has been removed. If central extension is found, intracranial removal of the entire nerve is advisable. To minimize the chance of ensuing meningitis, this should be postponed until complete healing has occurred after the orbital operation. Experience shows that meningitis frequently occurs when craniotomy and enucleation are performed too close together. Even without subsequent craniotomy, the danger of meningitis following the orbital approach is noteworthy. Verhoeff [2] observed that meningitis occurred in about 10 per cent of Hudson's cases when the orbital approach was the only procedure used. The danger appeared particularly great with intracranial involvement of the nerve, for in 10 of the 11 cases of meningitis such intracranial extension was found at autopsy.

As has been mentioned by Verhoeff,[2] in intraorbital operations it seems advisable to remove the eye with the tumor. This permits use of an artificial eye and affords a satisfactory cosmetic result. When the

tumor is removed without the globe, there may result an unsightly strabismus or ptosis, and, because of degenerative changes, the eye may ultimately require removal. However, Knapp [21] and Weskamp [22] have advocated preservation of the eye following a Krönlein procedure for removal of an intraorbital glioma. Knapp [23] says that 4 patients operated on by him by the Krönlein method have been followed now for fifteen to twenty years, and the eye remains perfectly normal in appearance and no intracranial disturbance has developed, in spite of the fact that 3 of these patients showed roentgen evidence of enlargement of the optic canal. Weskamp reported a case in which one and a half years after this method was used the patient's eye was in excellent condition in spite of sectioning of the central vessels and ciliary nerves.

Because complete removal of the glioma of the optic nerve with sufficient normal nerve affords the greatest chance of cure, in a healthy young person the intracranial procedure with unroofing of the orbit and section of the nerve distally and proximally to the globe in many ways seems the procedure of choice. Adequate dural closure prevents leakage of the cerebrospinal fluid and, from the standpoint of meningitis, makes subsequent enucleation less precarious.

Although the intracranial procedure is more hazardous from an operative point of view, in experienced hands the mortality rate is not high. Meningitis is less likely to occur than with the orbital approach, and much more certain determination of the extent of the neoplastic growth can be obtained. Some of the intraorbital gliomas of the optic nerve even extend so close to the optic foramen that complete intra-orbital removal is impossible.

PROGNOSIS

The prognosis for these neoplasms depends on the location and extent of the growth as well as on the degree of malignancy. After complete removal of an intraorbital tumor, the possibility of recurrence in the orbit is remote. DeLong [3] stated that recurrence under such circumstances has never been reported.

In view of the fact that in many cases removal has been incomplete and yet no case has been reported in which there has been recurrence of the tumor in the orbit, Verhoeff [2] suggested that perhaps excision of the largest part of the tumor might in some way eliminate a stimulating factor or substance, thus inhibiting further extension of the growth. Experience with similar types of cerebral tumors fails to support such a supposition. Moreover, the rarity of glioma of the optic nerve scarcely

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21. Knapp, A.: Contributions to Ophthalmic Science, Menasha, Wis., George Banta Publishing Company, 1926.

22. Weskamp, C.: Glioma of the Optic Nerve: Report of a Case, Arch. Ophth. **13**:630 (April) 1935.

23. Knapp, A.: Personal communication to the author.

allows a proper evaluation of this point of view. The fact that some of these tumors, as in the case here reported, have grown fairly rapidly within a period of several months makes one realize that incomplete removal would be hazardous and unjustifiable even though, in contrast to retinal gliomas, recurrence would be slow. Furthermore, although a partially extirpated tumor may not recur in the orbital tissues, there is no evidence that subsequent slow extension along the nerve to the brain is inhibited by partial removal.

Should the tumor involve the chiasm, there is little to be accomplished surgically or otherwise. Three of the 7 persons operated on by Dr. Cushing [8] died within forty-eight hours after operation, and in no instance was any beneficial result obtained. As mentioned by Grinker,[9] death with hyperpyrexia not infrequently occurs from manipulation of the chiasmal gliomas, probably as a result of disturbance of the heat-regulating center at the base of the brain behind the chiasm. Mehney's [24] recent report of 3 cases of glioma of the optic nerve from Peet's clinic included 1 in which the growth had extended into the chiasm. The patient died twenty-four hours postoperatively with hyperpyrexia.

It was the opinion of Martin and Cushing [8] that should clinical diagnosis of extension into the chiasm be sufficiently perfected, there would be every reason to avoid operation, although in case of doubt exploration would have to be carried out. High voltage roentgen therapy has been tried in these cases but has proved of little if any significant benefit.

SUMMARY AND CONCLUSIONS

Glioma of the optic nerve is rare, less than 300 cases having been reported. Seventy-five per cent occur in the first decade of life. In a high proportion of cases there is extension of the tumor into the cranial cavity.

A case is presented with the clinical and pathologic observations.

The suggestive diagnostic signs are the ophthalmoscopic appearance of atrophy of the optic nerve or optic neuritis, slowly progressive nonpulsatile exophthalmos and enlargement of the optic foramen, as seen roentgenographically.

The polar spongioblastoma is the invariable type of glioma found involving primarily the optic nerve. Since, from an embryologic standpoint, the optic nerve contains all the adult forms of neuroglia, it is logical to presume that advanced glial tumors will be reported in the future as more of these neoplasms are discovered and studied with differential staining methods.

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24. Mehney, G. H.: Primary Tumor of the Optic Nerve: Report of Three Cases, Arch. Ophth. **16**:95 (July) 1936.

Intracranial extension of glioma of the optic nerve occurs more frequently than is usually appreciated, as judged by the prevailing attitude of the ophthalmic literature regarding treatment of these growths. The unjustifiable hazard involved when the surgeon is content with incomplete orbital removal is not sufficiently realized.

A glioma of the optic nerve usually grows slowly, and orbital recurrence, even after incomplete orbital removal, has not been reported. In spite of this fact, however, extension along the proximal nerve to the optic chiasm may occur and implies a fatal prognosis. If such extension is to be prevented, complete removal of the involved nerve is necessary. Technically, complete extirpation is more certain and meningitis less likely to occur if done through the intracranial approach. Considering the incidence of meningitis following the orbital approach, the intracranial approach cannot be considered a radical procedure, since it minimizes the possibility of meningitis.

Roentgenograms of the optic foramen should be made in all cases in which a tumor is suspected. The finding of an enlarged optic foramen is specific indication for intracranial section of the nerve with removal of the intraorbital portion of the nerve at the same time.

When there is clinical evidence of intracranial extension, early intraorbital removal of the involved nerve is recommended. If all the tumor tissue has been removed, further operation is not indicated. Should invasion of the stump by neoplastic tissue be found, intracranial section of the optic nerve at the chiasm and entire removal of the stump should be carried out.

THE PRODUCTION OF CORNEAL ULCERS IN THE RABBIT

ROBB McDONALD, M.D.*

AND

HORACE PETTIT, M.D.

Associate in Bacteriology, University of Pennsylvania

PHILADELPHIA

Previous attempts to produce a standardized corneal ulcer in the rabbit have been unsatisfactory. Bach [1] in 1894 was the first to attempt the production of a corneal ulcer for the purpose of evaluating specific treatment. He was not able to obtain uniformity, nor was Key [2] or Bursuk.[3] Brown and Pugh [4] finally succeeded in producing a fairly satisfactory ulcer by using a strain of staphylococcus which they found only after many trials. Most strains gave either too much inflammatory reaction or none at all. No mention is made in their paper of the production of hemolysin or pigment by their strain.

Strumia and Scarlett [5] in 1936 succeeded in obtaining a consistent keratoconjunctivitis for their study of the therapeutic effect of specific bacteriophage. They were able to get consistent infection only by giving intradermal injections of heat-killed staphylococci for three days previous to the injection of live organisms into the cornea. This

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* Fellow in Ophthalmology.

Read at the Section on Ophthalmology of the College of Physicians, Philadelphia, Dec. 15, 1938.

From the Department of Ophthalmology and the Department of Bacteriology, University of Pennsylvania.

1. Bach, L.: Experimental Investigations on the Staphylococcus Ulcer of the Cornea and Its Therapeutics, Cong. périod. internat. d'opht., Compt. rend. **8**:133, 1894.

2. Key, B. W.: Influence of Protein Therapy on Experimental Staphylococcic Infection of the Rabbit's Cornea, Am. J. Ophth. **9**:351, 1926.

3. Bursuk, G. G.: Therapeutic Immunization with Immunogen B (Staphylococcic Vaccine) in Experimental Ulcer, Vestnik oftal. **10**:517, 1937.

4. Brown, A. L., and Pugh, J.: Effect of Intraocular Concentration of Typhoid Antibodies on Experimental Corneal Ulcers, with Report of Seven Cases, Arch. Ophth. **16**:476 (Sept.) 1936.

5. Strumia, M. M., and Scarlett, H. W.: Effect of Bacterial Lysate on Staphylococcic Keratoconjunctivitis in Rabbits, Arch. Ophth. **15**:47 (Jan.) 1936.

procedure was based on the work of Julianelle and his co-workers.[6] The latter investigators, however, used scarification of the cornea in their experiments on sensitivity. Strumia and Scarlett found that injection into the cornea gave more consistent results. Julianelle and his co-workers observed that weekly instillations of an extract of staphylococci into the scarified area were necessary for at least four weeks before a reaction could be obtained. The short period of sensitization with heat-killed staphylococci, which was found by Strumia and Scarlett and confirmed by us, is explainable on the assumption that sufficient antibodies were stimulated to give an antigen-antibody reaction in the cornea and thus limit the infection to a corneal ulcer.

Circulating antibodies are not usually detectable before the sixth day. From a week to ten days is required before a guinea pig is sensitized to horse serum sufficiently to show the usual systemic manifestations of sensitivity. It may be that the corneal reaction is a delicate test for the presence of specific antibodies; further investigation of this is desirable. The formation of antibodies in general is dependent on the amount of antigen given and the frequency of the dose. Too much antigen may inhibit the reaction in certain instances.

Brown and Pugh reported that typhoid antibodies appeared in the blood in small quantities (a titer of from 1:5 to 1:20) within from twenty-four to forty-eight hours after intravenous injection of a suspension of 10,000,000 killed organisms. The titer rises from this time onward, and if no subsequent dose is given, reaches its height on about the sixteenth day. According to the authors, the ocular vessels in the normal eye are impermeable to agglutinins, and a relatively low titer of the aqueous (1:2 or less) persists even in the presence of a rising titer of the serum. The titer of the aqueous was increased, however, by aspiration of the anterior chamber.

The present investigation is an attempt to produce a corneal ulcer which would be accompanied by a minimum amount of conjunctival reaction but would still show staining with fluorescein and infiltration of the cornea for at least ten days. We were fortunate to be able to obtain the original strain of staphylococci used in the experiments of Drs. Strumia and Scarlett.

EXPERIMENTAL METHOD

A six hour subculture of this original strain in neopeptone broth was used for the first corneal injection. On the following day material was obtained from the right eye of the rabbit and spread on a 5 per cent rabbit blood agar plate. After

6. Julianelle, L. A.; Morris, M. C., and Harrison, R. W.: An Experimental Study of Corneal Vascularization, Am. J. Ophth. **16**:962, 1933.

twenty-four hours at 37 C. the plate showed two types of colonies; one was yellow and hemolytic and the other, white and nonhemolytic. Each type was spread separately on a 5 per cent blood agar plate for isolation. Staining of both cultures with Gram's stain showed pure colonies of gram-positive cocci in clusters. After eighteen hours' growth at 37 C. subcultures of the albus strain were made in 0.5 per cent dextrose neopeptone broth. The albus strain was chosen for further work, because the aureus strain produced too much reaction. It is well known that Staphylococcus aureus haemolyticus is much more pathogenic [7] than the nonhemolytic albus strains. These were centrifuged after eighteen hours' incubation, the supernatant fluid was discarded and the sediment was suspended in 0.85 per cent solution of sodium chloride and heated at 60 C. for forty minutes. One loopful planted in broth showed no growth in eighteen hours. This suspension containing 4,000,000 heat-killed staphylococci per cubic centimeter was used for subsequent subcutaneous injection. It was stored without preservative in a refrigerator in a vial stoppered with rubber; required amounts were withdrawn by needle and syringe under sterile precautions.

The amount of antigen used in our experiments, which was 0.1 cc. of the suspension of heat-killed white staphylococci given subcutaneously each day for three days, followed by the injection of living staphylococci into the cornea on the fourth day, is within the usual limits of ordinary experimental procedure. No tests were made on the blood to determine the presence of circulating antibodies.

The corneal injections were made with eighteen hour subcultures of the nonhemolytic white organisms in 0.5 per cent dextrose neopeptone broth. Each culture was examined by Gram's stain for purity before injection. Thirty rabbits (60 eyes) were used in this investigation. Anesthesia was obtained with 2 drops of a 0.5 per cent solution of pontocaine hydrochloride. A 26 gage short beveled needle was used, and 0.05 cc. of the broth culture was injected intracorneally about 2 mm. from the limbus, care being taken to keep the point of the needle from entering the anterior chamber. Occasionally some of the culture leaked out around the needle, but this did not seem to affect the final result.

The animals were observed for the following criteria: secretion, corneal infiltrate, staining, hypopyon and the formation and regression of pannus.

RESULTS

The most constant factor seen in the animals was the appearance of pannus on the fourth or the fifth day. It usually appeared as a heavy brush of vessels from the limbus nearest the site of the ulcer; occasionally it appeared as a typical "salmon patch." This is without doubt the pannus of infection described by Julianelle and his co-workers, as it was definitely related to the severity of the infection. The ulcer did not begin to improve until the pannus appeared.

The ulcers produced were accompanied by a moderate conjunctival reaction and secretion. This reaction subsided gradually after the third day and was the type that might be expected with any pyogenic corneal ulcer. The cornea was hazy within twenty-four hours, the

7. Chapman, G. H.; Berens, C.; Nilson, E. L., and Curcio, L. G.: Differentiation of Pathogenic Staphylococci from Nonpathogenic Types, J. Bact. **35**:311, 1938.

hazing increasing slightly till about the third day and then remaining about the same until the ulcer began to heal on about the seventh or the eighth day. Staining with fluorescein was present until the haze had practically disappeared. The ulcers usually measured from 2 to 3 mm. in diameter and involved the superficial layers of the substantia propria. They healed completely, leaving only a faint nebula and empty vessels at the end of three weeks. When the opposite eyes of the first 12 rabbits were inoculated about three weeks after the original injection, no difference in the duration and severity of the ulcers from that in the first eyes was noted. The most uniform results were obtained when both eyes of 8 rabbits were inoculated at the same time. The severity of the infection varied to some extent for each individual of the species, but there was marked uniformity in the appearance and course of the two eyes when they were inoculated at the same time. Six rabbits that were not sensitized to specific vaccine showed more variation than those that had been sensitized, though the two eyes of each animal were remarkably similar. The variation observed in the nonsensitized rabbits was either toward more extensive involvement of the eye or toward a minimal reaction; approximately half of the eyes cleared completely in from three to four days without treatment, while in 2 of the rabbits hypopyon ulcers developed accompanied by a fairly severe reaction. The 8 eyes into which dead organisms were injected showed slight conjunctival reaction in twenty-four hours; the cornea of each eye was clear but stained at the site of the injection. Within forty-eight hours all that remained was a faint nebula, which practically disappeared in four days. No pannus was observed in any of these rabbits.

Hypopyon ulcers were produced in 4 eyes. This was a complication that we tried to avoid. It occurred early in our series when we were perhaps less skilful in our technic and twice in the last series when the animals were not sensitized.

Some of the ulcers were treated when we observed that a consistent ulcer was being obtained. Five eyes were irradiated with grenz rays (in varying doses). We realized at the time that this type of treatment might not be satisfactory, since the ulcers were fairly deep. The results in this small series were not significant. Sixteen eyes were treated with subconjunctival injections of Pregl's solution (a complex aqueous solution containing 3 per cent of available iodine); 0.5 cc. was injected every day for three days, starting twenty-four hours after the corneal injections. Though every ulcer did not respond to this treatment, the average duration of secretion, hazing and staining was noticeably less than in the eyes that were not treated (fig. 2).

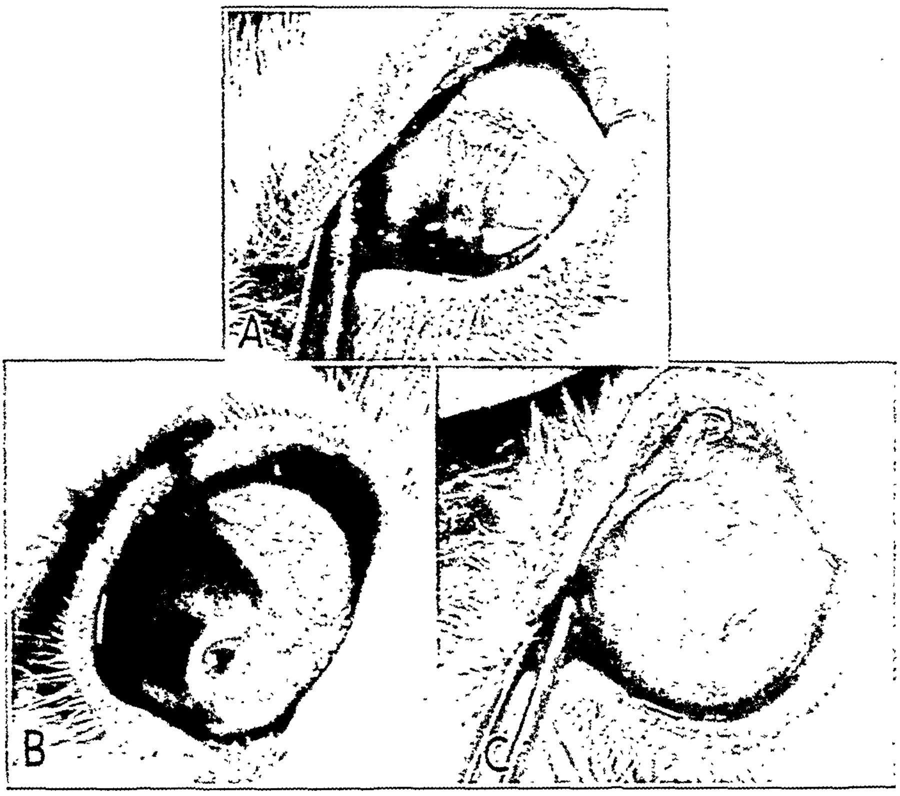

Fig. 1.—*A*, typical pannus formation extending to the site of the ulcer. *B*, ulcer after staining with fluorescein. The staining slightly exaggerates the loss of substance. *C*, early pannus forming at the limbus, hazing of the cornea and circumscribed ulcer.

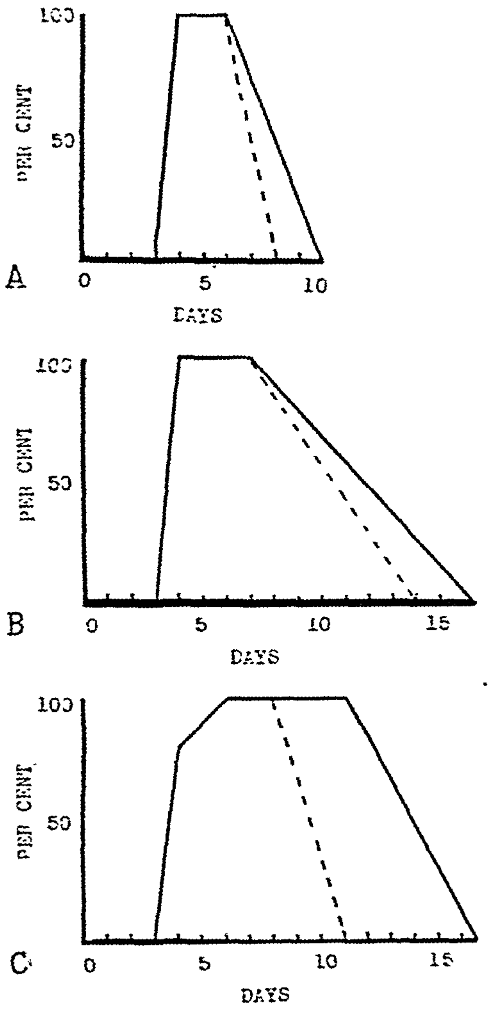

Fig. 2.—*A*, duration and course of corneal secretion. *B*, duration and course of staining of the cornea. *C*, duration and course of corneal infiltration. The ordinates represent the percentage of involvement; i. e., 100 per cent equals maximum reaction. The abscissas represent the time in days. The corneal injections were made after three days of sensitization to a specific vaccine. The dotted lines represent the course of the reaction in the treated animals as compared to that in the animals that were not treated (solid lines).

COMMENT

We feel that a fairly standard staphylococcic ulcer suitable for experimental purposes can be obtained, providing one uses rabbits of approximately the same size and weight and sensitizes them with a vaccine of the same strain that is used to produce the ulcer. When one uses a nonhemolytic nonpigment-forming strain of staphylococci, one can obtain an ulcer that has a minimum amount of conjunctival reaction but will show infiltration and staining of the cornea for approximately from ten to twelve days. When injections are made into both eyes at the same time, there is marked uniformity in the appearance and course of the two ulcers. We suggest that this procedure be carried out when one eye is to be treated and the other used as a control.

SUMMARY

Standard corneal ulcers were produced by injecting living Staph. albus into the corneas of rabbits previously sensitized by subcutaneous injection of specific heat-killed vaccine.

CONGENITAL FAMILIAL EXTERNAL OPHTHALMOPLEGIA WITHOUT PTOSIS

WITH A LESION OF THE PYRAMIDAL TRACT

MAX HELFAND, M.D.
NEW YORK

Congenital disturbances of the ocular muscles occur in the following order of frequency: (*a*) ptosis, (*b*) paralysis of the abducens nerve with or without involvement of the facial nerve and (*c*) complete or incomplete involvement of the third nerve with ptosis. Congenital familial external ophthalmoplegia without ptosis is rare.[1] A case of this anomaly associated with involvement of the right side of the pyramidal tract is reported. The interest in this case lies not only in the rarity of the condition but in the clinical data, which point to a better understanding of the etiology of such anomalies.

A. H., a woman aged 22, was admitted to the neurologic clinic of the New York Post-Graduate Medical School and Hospital complaining of "nervousness," a feeling of tension and "generalized fatigability."

The family history revealed that the patient's grandmother was supposed to have been afflicted with "eye trouble." Persons would remark that she constantly moved her head from side to side when she wanted to observe an object. She could not move her eyes voluntarily. The patient's sister also had similar trouble with her eyes. She could not move them; they were "absolutely stationary." However, she died of sarcoma of the stomach at the age of 21.

The patient's past history was essentially unimportant. Her birth was normal, and she was breast fed. She began to walk at 1 year of age and to talk at 16 months. She had measles at 2 years of age and scarlet fever at 6. Her mother, whose intelligence was limited, stated that she noticed the patient's eyes did not turn when the child was about 2 years of age.

On examination the patient seemed alert and bright looking but had a fixed stare. Her head moved about constantly in all directions, giving one the impression of a restless wandering movement. Her gait was normal, and the associated movements were within normal limits. There were fine horizontal tremors of the outstretched hands; they were not accentuated on intention. The muscular system throughout the body revealed normal strength, and there was no atrophy and no fibrillation. All the deep reflexes of the upper and lower extremities were equal but slightly hyperactive. The abdominal reflexes, both the upper and

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From the neurologic clinic of the New York Post-Graduate Medical School and Hospital.

1. Ulrich, O., in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1933, vol. 16, p. 145.

the lower, were present but unequal; those on the right were diminished. There was an equivocal Babinski sign on the right and positive Oppenheim and Gordon signs on the right. The sensory examination for all modalities revealed normal findings. The ocular examination, which was checked in the ophthalmic department of the hospital, revealed the following data: Vision was 21/100 in the right eye and 20/40 in the left eye. The levator palpebrae muscles showed good function. When the patient attempted to look upward she overacted the frontalis muscles, but her eyes did not move. There was bilateral ophthalmoplegia externa. Slight bilateral external rotation was retained. The pupils were equal and reacted to light and in accommodation. There was no convergence. There was a constant oscillation of the eyes as seen with the ophthalmoscope. All the other cranial nerves were normal.

Roentgen examination of the skull showed no gross abnormality in the detail of the outline of the bones of the vault or of the base. The bones of the vault were within normal limits of developmental variation. There was perhaps an increase in the structural density along the left supraorbital ridge.

The sella turcica was slightly small but nevertheless was within 10 per cent of the normal developmental variation. It was smooth in contour, with no discernible evidence of erosion or pressure atrophy. Neither was there any evidence of such changes elsewhere.

There were small calcific flecks in the region of the frontal bones in the midline.

The accessory sinuses and mastoids showed fair transillumination.

The Wassermann reaction was negative, and the basal metabolic rate was average normal.

COMMENT

Various theories have been advanced to explain these anomalies. The circulatory theory [2] ascribed the etiologic cause to a hemorrhage in the sheaths of the orbital nerves or in the brain itself. This insult most usually occurs during birth trauma. The neurogenic theory [3] was first advocated by Mobius. This placed the lesion centrally as a slowly degenerative process in the nuclei of the motor nerves of the eye. Mobius thought it to be an infantile nuclear atrophy. This, of course, presupposed the presence of an influencing factor in the nuclei which caused it gradually to become atrophic. This explanation was further strengthened by the fact that minimal excursions were always present.[4] With further study of these anomalies and with reports of postmortem

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2. Oppenheim, H.: Text-Book of Nervous Diseases, ed. 5, translated by A. Bruce, New York, G. E. Stechart & Co., 1911, vol. 2, p. 1041.

3. Bernheimer, S.: Aetiologie und pathologischen Anatomie der Augenmuskellähmungen, in von Graefe, A., and Saemisch, T.: Handbuch der gesamten Augenheilkunde, Leipzig, Wilhelm Engelmann, 1907, p. 34. Langdon, H. W., and Cadwalader, W. B.: Chronic Progressive External Ophthalmoplegia, Brain **51**:321, 1928. Mobius, P. J.: Ueber infantilen Kernschwund, München. med. Wchnschr. **39**:17 (Jan. 12) 1892.

4. Li, T. M.: Congenital Total Bilateral Ophthalmoplegia, Am. J. Ophth. **6**:817, 1928.

observations,[5] the conception of the nature of the lesion varied. Autopsy did not disclose any degenerative process but merely a defect in the number of cells in the nuclei as well as reduced neural elements in the other motor systems, such as the pyramidal tract, the substantia reticulata and the olives. The fact that patients with ocular anomalies presented other structural abnormalities of the body also added difficulty to the acceptance of the theory of infantile nuclear atrophy.

The muscular theory [6] advocated primary atrophy of the muscles in the eye with secondary retrograde degeneration in the nuclei. Fuchs [7] excised pieces of the levator palpebrae muscle and found thinning of the fibers and degeneration of the sarcolemma. This theory could not adequately explain the ocular defect, so de Schweinitz [8] was led to attribute it to a combination of nuclear defects with a developmental defect in the muscle. Zappert [9] attributed the cause to traumatic injury at birth of the nerves which supply the ocular muscles, with retrograde degeneration of the nuclei.

While some authors have attempted to rationalize along Vogt's theory of pathoclisis [10] or the theory of defects of the segmental anlage,[11] the most important contribution to this question has been derived from studies in embryology. The embryopathophysiologic theory explains the ocular disturbances in these cases. It not only is supported by clinical data but can be verified by experimental procedures. By exposing a definite mouse strain to roentgen irradiation, Bagg [12] produced anomalies of the eyes and the extremities. These were described by him as being

5. Spatz, H., and Ullrich, O.: Klinischer und anatomischer Beitrag zu den angenborenen Beweglichkeitsdefekten im Hirnnervenbereich, Ztschr. f. Kinderh. **51**:579, 1931.

6. Gourfein, D.: Un cas de double ophthalmoplegie extérieure congénitale et hereditaire chez six membres de la même famille, Rev. méd. de la Suisse Rom. **16**:673, 1896. Bradburne, A. A.: Hereditary Ophthalmoplegia in Five Generations, Tr. Ophth. Soc. U. Kingdom **32**:142, 1912. Lawford, J. B.: Congenital Hereditary Defect of Ocular Movements, ibid. **8**:262, 1888.

7. Fuchs, E.: Ueber isolierte doppelseitige Ptosis, Arch. f. Ophth. **36**:234, 1890.

8. de Schweinitz, G. E.: Complete Bilateral Congenital Exterior Ophthalmoplegia and Double Ptosis; Bilateral Cerebral Cortical Atrophy of the Frontal and Parietal Regions (Encephalograms); Clinical Communication, Arch. Ophth. **5**:15 (Jan.) 1931.

9. Zappert, J.: Ueber infantilen Kernschwund, Ergebn. d. inn. Med. u. Kinderh. **5**:305, 1910.

10. Vogt, O.: Der Begriff der Pathoklise, J. f. Psychol. u. Neurol. **31**:245, 1925.

11. Bauman, G. I.: Absence of the Cervical Spine: Klippel-Feil Syndrome, J. A. M. A. **98**:129 (Jan. 9) 1932.

12. Bagg, H. J.: Disturbances in Mammalian Development Produced by Radium Emanation, Am. J. Anat. **30**:133, 1922.

recessively inherited. They were produced by the formation of a bleb at the anlage of the eyes and of the extremities. The ocular abnormalities represented a constant finding in all of the experiments. Bagg and Little [13] continued these experiments and found the origin of these blebs. They showed that in all embryos of about 8 mm. a clear fluid is expelled through an anterior foramen situated on the roof of the myelencephalon. This fluid is expelled in order to release the internal pressure of the medullary tube, as the latter is in the process of folding on itself.[14] The anterior foramen exists for a short time, between 7 and 10 mm. of embryonic growth. It is then pulled into the anterior part of the fourth ventricle. Its function is taken over by the foramen Magendi, which is formed posterior to the choroidal plexus.

The expressed clear fluid lies on the dorsal surface underneath the integument. Normally, the fluid is driven along mechanically by the elasticity of the epidermis along the concavity of the surface. When there is an abnormal amount of fluid or when there is a hindrance in its path and the bleb is enlarged, there is interference in the embryonic anlage. A prolonged delay at the flexure or ridge may cause an irritation of the capillary bed, and the bleb may be filled with blood. Bennevi [15] stated that "in living embryos I have often observed blood capillaries to burst at the bottom of a clear bleb, and pour the bloody contents into the cellular bleb." The clear bleb and occasionally the bloody bleb may be absorbed, but the abnormally delayed bleb produces typical abnormalities of the eyes and the extremities.

The nuclei of the motor nerves to the eye are all situated at the crucial encephalic flexure in the human embryo. At 7 mm. of embryonic growth the end of the neural tube is bent sharply in the region of the midbrain, so that the axis of the forebrain forms a right angle with the axis of the hindbrain. As the brain bends, the intramedullary canal becomes constricted into the isthmus, thus increasing its inner pressure.[16] It is at this point that an abnormally large bleb may be retained and later filled with blood. The pressure of the bleb on the relatively superficial nuclei produces the defect in the anlage of those tissues.

The foregoing phenomenon explains the occurrence of complete and partial ophthalmoplegia. The nucleus of the third nerve is made up of

13. Bagg, H. J., and Little, C. C.: Hereditary Structural Defects in the Descendants of Mice Exposed to Roentgen-Ray Irradiation, Am. J. Anat. **33**:119, 1924.

14. Keegan, J. J.: Comparative Study of the Roof of the Fourth Ventricle, Anat. Rec. **2**:379, 1917.

15. Bennevi, K.: Embryological Analysis of Gene Manifestations in Little and Bagg's Abnormal Mouse Tribe, J. Exper. Zoöl. **67**:443, 1934.

16. Weed, L. H.: The Development of the Cerebrospinal Spaces in Pig and in Man, Publication 225, Carnegie Institution of Washington, 1917.

groups of ganglion cells extending along the axis of the aqueduct. An injury to any of the groups of these cells might produce a change in the clinical symptoms. It is interesting to note that the nuclei of the levator muscles of the eyelids are placed most cephalad. A lesion which does not extend the entire length of the nuclei would spare the function of these muscles.

The involvement of the right side of the pyramidal tract in the case reported becomes intelligible in the light of the experiments of Bagg and Little. These authors constantly found the occurrence of ocular anomalies together with deformities of the extremities, for the abnormal bleb was usually retained on the dorsal side of the organism and nearly always around the anlage of the eyes and the extremities. It is also plausible that a mechanical hindrance in the form of an abnormal bleb situated at the cephalic flexure might injure the anlage of the motor tract and spare the sensory system. These interferences usually occur in embryos of about 7 or 8 mm., or at the fourth or fifth week of embryonic life, at which time the sensory anlage is already ripened and is therefore more resistant to pressure.

SUMMARY

A case of congenital familial external ophthalmoplegia without ptosis and with signs referable to involvement of the pyramidal tract has been described.

Various theories to account for these anomalies have been studied.

The pathophysiologic disturbance in embryonic development fully explains such findings.

BILATERAL IRITIS COMPLICATING SERUM SICKNESS

FREDERICK H. THEODORE, M.D.

AND

ARTHUR C. LEWSON, M.D.

NEW YORK

The usual manifestations of serum sickness are common and are universally recognized. Even relapses, which occur from time to time after free intervals, are well known. Focal reactions in the course of serum sickness, however, are much rarer. The most common type is paralysis due to neurologic disturbances, of which about 100 cases have been reported.[1] Ocular involvement in serum sickness has apparently been the subject of only three contributions, two of which deal with optic neuritis and the third with edema of the retina.[2] The first was that of Mason,[3] who in 1922 described 3 cases in which measurable blurring of the optic disks developed during serum sickness. Two of the patients had received antipneumococcus serum, while the third had been given antimeningococcus serum. A gradual return to normal occurred. In another report Brown [4] in 1925 observed that of 75 persons receiving diphtheria antitoxin, a measurable elevation of the disks and edema of the retina developed in about 20 per cent in from two to three hours after administration of the serum. This subsided shortly, only to return a week later when serum sickness had developed. The amount of involvement of the optic nerve, which in 10 per cent of all cases caused visual disturbances, paralleled the intensity of the general reaction to the serum. The third report was that of Bedell,[5] who in 1935 described severe and extensive bilateral retinal edema with

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1. Neffson, A. H.: Recurrent Paralysis of the Larynx Following Injection of Tetanus Antitoxin, Arch. Otolaryng. **27**:201-203 (Feb.) 1938.

2. We have not included the case of Walker (Anaphylactic Keratitis, J. A. M. A. **80**:160-161 [Jan. 20] 1923) in which severe keratitis developed two days after the intravenous injection of a "serum" made from the patient's own blood, which caused an immediate anaphylactic reaction.

3. Mason, V. R.: Optic Neuritis in Serum Sickness, J. A. M. A. **78**:88-89 (Jan. 14) 1922.

4. Brown, A. L.: Ocular Manifestations in Serum Sickness, Am. J. Ophth. **8**: 614-618 (Aug.) 1925.

5. Bedell, A. J.: Stereoscopic Fundus Photography, J. A. M. A. **105**:1502-1509 (Nov. 9) 1935.

superficial hemorrhages, which he attributed to serum sickness following the injection of tetanus antitoxin. This cleared up rapidly, leaving few vestiges. Although experimental work indicates the vulnerability of the iris in allergic reactions, apparently no case in which iritis occurred in the course of serum sickness has ever been recorded. Because of this, it was thought worth while to report the following case.

REPORT OF CASE

B. H., a 40 year old salesman, was first seen by one of us (A. C. L.) on March 30, 1938. He had had a productive cough for one week and complained of chills, fever and pain in the chest of twelve hours' duration. The past history was of no importance. No allergic history was elicited. The diagnosis of lobar pneumonia was made despite the absence of signs referable to the chest other than restricted breathing due to pain. The patient was admitted that night to the Beth Israel Hospital, where the diagnosis was confirmed by roentgen examination, which showed involvement of the right middle and lower lobes. The sputum revealed type I of the pneumococcus, and after both intradermal and ocular sensitivity tests to horse serum were found to be negative, Lederle's type I antipneumococcus serum was immediately administered intravenously, 20,000 units at a time, with 1 cc. of epinephrine hydrochloride (1 : 1,000) subcutaneously. After the third dose of serum, the patient had a moderately severe anaphylactic shock characterized by restlessness, pain in the back, pallor and sweating. After he had recovered from this, a fourth and last dose of serum diluted with 5 per cent dextrose in physiologic solution of sodium chloride by intravenous drip caused no reaction. A total of 80,000 units was given, all on March 31. That night some transient hives were noted.

After the administration of the serum the temperature dropped in two days from a maximum of 105 F. to 101 F., coincidentally with a marked improvement in the patient's general condition. His course was complicated, however, by a pleural effusion, first noted on April 7, which prolonged convalescence considerably, lasting till April 21.

On April 8, nine days after the administration of the serum, there developed serum sickness characterized by an elevation of temperature to 102.4 F., pruritus, urticaria and generalized lymphadenopathy. The spleen was not palpable. The eyes at this time showed no abnormality. The serum sickness lasted two days. After April 10 the temperature was never above 99.4 F., and the patient was discharged from the hospital on April 17. No significant laboratory findings other than those mentioned were noted during his stay in the hospital.

On April 30 serum sickness again developed, manifested this time by an elevation of temperature to 101 F., pains in almost every joint and some transient urticaria. Along with these symptoms the patient suddenly began to note photophobia accompanied by slight pain in each eye and then blurring of vision. There was considerable tearing and some mucopurulent discharge. As all the symptoms continued, he consulted his physician (A. C. L.) on May 3, who prescribed ephedrine sulfate by mouth and drops of epinephrine hydrochloride (1 : 1,000) for the eyes. The fever and arthritic symptoms abated in a few days, but because there had not been much ocular improvement other than diminution in the discharge, the patient consulted an ophthalmologist (F. H. T.) on May 7.

At the time of ocular examination the presenting symptoms were photophobia and impairment of vision, which, although originally equal in each eye, were now

less marked in the left eye. Little, if any, pain was present. The vision in the right eye was 20/70 and could not be improved with glasses. The vision in the left eye, which was 20/40 + 1, also could not be improved. The preauricular gland on each side was moderately enlarged. The palpebral conjunctiva was injected to a considerable degree in the right eye and slightly in the left eye. A small amount of mucoid discharge was noted. No organisms were found on staining with the Gram stain. The Wright stain showed numerous pus and epithelial cells but no eosinophils or inclusion bodies.

Moderate circumcorneal injection was present in the right eye. On slit lamp examination the corneal epithelium, which did not stain with fluorescein, showed bedewing. Descemet's membrane showed numerous folds. The endothelium on its posterior surface was covered with so much fluffy white material that it appeared as if a piece of cotton wool were covering the entire layer. This deposit precluded further microscopic examination of the interior of the eye, but the anterior chamber was seen to contain much similar cotton-like exudate. The pupil appeared only slightly contracted. The media and fundus could not be examined. The ocular tension was slightly diminished on palpation.

Only slight circumcorneal injection was present in the left eye, but a fibrinous iritis, similar to that in the right eye but less intense, was seen. Much cotton-like exudate was noted both in the anterior chamber and on the endothelium. The pupil was slightly contracted. No abnormalities were found in the lens, vitreous or fundus. The ocular tension appeared normal on palpation.

The most striking thing about the patient was the relative lack of discomfort and lack of reaction in the face of such an extensive process. Because of this, treatment was limited to the instillation of a 1 per cent solution of atropine sulfate three times a day in the right eye and once a day in the left eye in addition to the use of hot compresses every three hours. The improvement was remarkable. The photophobia quickly disappeared; in six days the vision in each eye was 20/20. Within three days a tremendous amount of absorption of the exudate occurred; in the course of a week practically none remained except as small deposits on the endothelium. These, too, disappeared in another ten days. Careful study of the fundi for possible choroidal foci at this time gave negative results.

Studies for focal sources of the iritis, including dental and otolaryngologic examinations, revealed no significant findings. The Wassermann reaction of the blood was negative. A blood smear on May 7 showed no eosinophilia or abnormal proportion of lymphocytes.

COMMENT

Ever since the first experimental investigations into the nature of allergy, the eye has been utilized because of the great advantages it offers for such studies. Horse serum has been used as an antigen in many of these experiments. It was discovered comparatively early by the works of Nicolle and Abt, Sattler, Krusius, Romer and Gebb, Kümmell, von Szily and Arisawa, Zade, Morax [6] and others that allergic ocular inflammatory reactions could be produced by a number of

6. Cited by (*a*) von Szily, A.: Die Anaphylaxie in der Augenheilkunde, Stuttgart, Ferdinand Enke, 1914, chap. 7; (*b*) Woods, A. C.: Ocular Anaphylaxis: I. The Reaction to Perfusion with Specific Antigen, Arch. Ophth. **45**:557-573 (Nov.) 1916.

methods. In all their experiments, however, at some time or other intraocular injection was performed. In 1916 Woods,[6b] using horse serum as one antigen, showed that in dogs sensitized by intraperitoneal injections without injury to the eye a constriction of the pupils occurred when the heads were perfused later with the specific antigen. Small retinal hemorrhages also occurred. In later studies Woods[7] showed that in dogs sensitized by intraocular injections of an emulsion of uveal pigment later injections intraperitoneally caused allergic iridocyclitis in the other eye. Somewhat similar findings had been noted by Dold and Rados.[8] Riehm[9] confirmed this in 1930, using horse serum. All these later experiments were done in studies of sympathetic ophthalmia but are cited here to indicate that the iris responds readily to an allergic inflammation.

In 1930 Iga[10] published a report that is of great interest in connection with the case reported here. His attention had been drawn to focal inflammatory reactions that occurred in the normal eyes of animals in the course of immunization with horse serum. He thereupon began investigations to study the effects on normal eyes of various foreign serums injected parenterally. At no time was the eye itself touched. Iga found that single intravenous injections of even such small amounts of horse serum as 0.5 cc. could produce a diffuse or nodular lymphocytic infiltration of the uvea of both eyes, especially marked in the region of the choriocapillaris. The reaction was proportional to the amount injected; a definite cumulative effect was observed on repeated injections within the preanaphylactic phase, as in immunization. Definite clinical manifestations of uveal inflammation, such as pericorneal injection and clouding of the anterior chamber, with transient plastic iridocyclitis and large choroidal foci could be produced either on two or three injections of 10 cc. of horse serum or on the single injection of at least 20 cc. His work is well illustrated with drawings and photomicrographs. It was Iga's opinion that the single injection given could produce such a reaction because there was a primary toxic effect of foreign serum which, he assumed, acted

7. Woods, A. C.: Ocular Anaphylaxis: V. Experimental Iridocyclitis, Arch. Ophth. **47**:162-172 (March) 1918.

8. Dodd, H., and Rados, A.: Versuche über sympathische spezifische und unspezifische Sensibilisierung, Ztschr. f. Immunitätsforsch. u. exper. Therap. **20**: 273-291 (Dec.) 1916.

9. Riehm, W.: Ueber die Bedeutung der Anaphylaxie für den klinischen Ablauf der sympathischen Ophthalmie, der Tuberkulose und der organgebundenen Infektionskrankheiten (Nach Studium am Auge), Arch. f. Ophth. **123**:361-426, 1930.

10. Iga, F.: Ueber Herdreaktionen an den unberührten Augen nach parenteraler Zufuhr von artfremden Serum, Klin. Monatsbl. f. Augenh. **84**:449-478 (April) 1930.

as a capillary poison. He found that the eye, because of the richly vascular uvea, and the lungs, the liver and the brain and meninges were usually affected.

Thus, the vulnerability of the animal iris to the effects of foreign serum appears to have been adequately proved experimentally. It is surprising in view of this that no clinical cases have been noted until the present time. When one considers the protean manifestations of serum sickness, there seems no reason that it should not occasionally include iritis. In the case here reported all the evidence points to such an etiology. Since the same shock organ, in this case the uvea, is present bilaterally, it follows that both eyes were simultaneously affected; at the same time the other symptoms began. The entire picture suggested a transitory exudative reaction into the anterior chamber, much like that in other synovial cavities, like those of joints. From the beginning it was felt that we were dealing with an unusual sort of process because of the disparity between symptoms and findings. It caused little distress and cleared promptly in the course of a week, so unlike the usual iritis with equally extensive pathologic changes. Even the large amount of exudate disappeared soon. The experimental iritis that Iga produced with horse serum likewise proved mild and transitory. Therefore, while one can never be absolutely certain on clinical grounds as to the etiology of a condition such as iritis, we feel that in this case it was a manifestation of serum sickness.

STREAK RETINOSCOPY

WALTER Z. RUNDLES, M.D.
FLINT, MICH.

Refraction is a procedure of major importance in the practice of ophthalmology. The majority of all patients who consult the ophthalmologist do so because of refractive errors the proper correction of which relieves them of symptoms of ocular distress. It is well known, too, that refractive errors may be etiologically important when symptoms seem to indicate extraocular or intraocular disease. Thus in numerous instances symptoms of apparent muscle imbalance, psychogenic disorder or involvement of the central nervous system can be correctly evaluated only after existing refractive conditions have been thoroughly considered.

The numerous and diverse methods used for refraction may be divided into two general groups: the subjective and the objective. The first involves the use of visual acuity charts, trial lenses etc., and depends on the patient's judgment of his visual capacity with different lens combinations. Objective methods of refraction employ such instruments as the retinoscope and the ophthalmometer and are relatively independent of the patient's visual processes. Although many attempts have been made to emphasize one type of test to the practical exclusion of the other, such is not the present intention. I am in full accord with the prevailing opinion that accurate refraction depends on the skilful use of both types of examination with adaptations appropriate to the individual case. The value of objective methods in routine ocular examination is entirely contingent on the accuracy and reliability of the information obtained. The final criterion by which objective methods are judged remains, of course, the subjective one of visual ability and ocular comfort. Not infrequently, however, subjective tests are found to be of limited utility, as in examining young children or adults of low intelligence, those who are grossly inaccurate in observation or who are unresponsive or antagonistic. Under these circumstances the refractive error is best estimated wholly by objective methods.

The retinoscope provides the most useful and practical means of estimating refractive errors objectively. The outstanding advantage of retinoscopic examination is that the total refractive error of all the dioptric media is measured. Plane mirror retinoscopy as developed

and described by Jackson [1] is virtually the universal method, with the self-luminous type of electric retinoscope the instrument generally preferred.

The purpose of this paper is to discuss another method of retinoscopy, that of streak retinoscopy. Although the streak retinoscope was invented many years ago, its outstanding merits have yet to be generally appreciated. More than a decade ago dissatisfaction with the accuracy and results of plane mirror retinoscopy led me to purchase an electric streak retinoscope.[2] By experimentation a procedure was developed suitable for practical refractive work. Daily use of this instrument for several years since then has provided convincing proof of the unique advantages in accuracy and convenience of this method of estimating refractive errors objectively.

HISTORY OF STREAK RETINOSCOPY

The streak retinoscope was invented in 1900 by Hugo Wolff, of Berlin, Germany. The instrument was designed to give a rectilinear band or streak of light for retinoscopy instead of the round spot of light provided by the plane mirror retinoscope. The optical principles and theory of retinoscopy (skiascopy) and the construction and practical use of his instrument were discussed at length by Wolff in a monograph published in 1903.[3] In later years the subject was further elaborated in a second monograph [4] and in a series of about eleven articles, the most significant of which are listed.[5]

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1. Jackson, E.: Skiascopy, in de Schweinitz, G. E.: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 120.

2. Reid streak retinoscope, J. E. Reid Instrument Co., Philadelphia.

3. Wolff, H.: Ueber die Skiaskopietheorie, skiaskopische Refractionsbestimmung und über mein elektrisches Skiaskopophthalmometer nebst Bemerkung über die Akkomodationslinie und die sphärische Abberation des Auges, Berlin, S. Karger, 1903.

4. Wolff, H.: Skiaskopietheorie vom Standpunkt der geometrischen Optik, der Ophthalmoskopie und entoptischen Wahrnehmung (entoptische Skiaskopietheorie), Berlin, S. Karger, 1905.

5. Wolff, H.: Ueber die Skiaskopietheorie und über mein elektrisches Skiaskopophthalmometer, Wien. med Wchnschr. **54**:2311-2315, 1904; De la théorie skiascopique et de mon skiascopophtalmomètre électrique, Arch. d'opht. **24**:213-219, 1904; Noch einmal meine Skiaskopietheorie, Arch. f. Augenh. **53**:135-148, 1905; Neue Beiträge zur Skiaskopietheorie, Wien. klin. Rundschau **20**:41-44 and 61-64, 1906; Das System der Skiaskopie und Ophthalmoskopie vom Standpunkt der physischen, physiologischen und geometrischen Optik, Ztschr. f. Augenh. **16**:91-195, 1906; Ueber Schattendrehung und Schattenlauf sowie über das astigmatische Gesichtsfeld in der Skiaskopie, Arch. f. Augenh. **60**:210-254, 1908; Vereinfachte Erörterung über Skiaskopie, nebst einer Uebersicht über 393 Untersuchungen, Ztschr. f. Augenh. **38**:318-359, 1917.

While much of Wolff's work is now only of historical interest, the construction of his instrument, his procedure for retinoscopy and his practical results may be briefly reviewed with profit. In the first streak retinoscope the source of illumination was a small, 12 volt electric bulb with a straight, horizontal rod filament. By means of a convex lens the image of the filament was projected onto a pierced plane mirror. The latter was mounted so that it could be rotated around an axis parallel to the source of light. When the mirror was rotated in this manner, the streak of light, i. e., the image of the straight filament, was turned through 180 degrees. In this way the eye was examined with the streak of light passing through any desired meridian.

The procedure which Wolff developed for objective refraction resembles in many ways that used today. Thus he emphasized the basic principle of the streak retinoscope, namely, that with this instrument the refraction is always to be determined in one meridian of the eye at a time—in the meridian lying at right angles to the streak. The "with" and "against" movements of the fundus reflex and the point of reversal were observed and interpreted as they are today. Both the stable method of retinoscopy at a fixed distance and the labile method with variable distance were used to measure the strength of correcting lenses. The axis of astigmatism was found in substantially the same manner as is subsequently described.

From the standpoint of practical utility, Wolff believed that his method transformed retinoscopy from a previously gross control method into one of the most exact ophthalmometric measuring methods. He insisted from his earliest work (1903) that the refraction of the eye, including the astigmatic correction, could by his method be estimated objectively to within 0.25 diopter, or even 0.1 diopter if one wished, and that the axis of the cylinder could be determined to an accuracy of 1 degree. The objective and subjective refractive estimations for 92 astigmatic eyes were tabulated for comparison to show that this accuracy was obtained in 80 per cent of the total cases. In the 20 per cent minority a greater accuracy was obtained than was possible with any other method. In Wolff's last paper (1917) the same conclusions were reaffirmed after seventeen years of experience with the method. The data on 393 cases were then tabulated to illustrate the accuracy of his procedure under various circumstances.

Although streak retinoscopy became well known throughout Germany due to Wolff's work, it did not attract a wide scientific following, perhaps because of the prevailing attitude regarding the medical importance of refraction itself. In other countries the method attracted even

less attention. In 1917 the distinguished British ophthalmologist R. H. Elliot commented as follows:[6]

> It is a good many years since Hugo Wolff brought out his electric retinoscope with a single line of light, and developed its technique in a monograph of truly Teutonic thoroughness, and yet his instrument is still almost unknown in Great Britian. I have used it now certainly for more than a decade, and I should indeed feel lost without it, so much so that I have tried to get an English instrument made on similar lines. . . . To my mind the advantages of the instrument cry aloud for recognition.

The principle of the streak retinoscope was later discovered independently in this country by J. C. Copeland.[7] While he was using a plane mirror retinoscope the frosted glass used to diffuse the image of the filament was accidentally broken. This caused the filament line to be projected instead of the conventional spot of light produced by the diffusing disk which covered a small aperture over the lamp. The filament image was S shaped, and the middle portion when projected over the pupil of the eye produced a straight line reflex. Further experimentation showed the value of the straight line reflex in retinoscopic work, and accordingly a retinoscope was designed to give this type of illumination. The instrument developed was patented in 1927 and accepted by the Council on Physical Therapy of the American Medical Association in 1935.[8] The illumination originates from a small bulb the filament of which is bent to provide a linear source of light. The bulb can be rotated 180 degrees as well as moved to and from a condensing lens to vary the angularity of the light leaving the mirror. At one extreme the rays converge to cross about 9 inches (22.8 cm.) in front of the instrument. At the other extreme the rays diverge, giving a "plano mirror effect." The use of this instrument was briefly described recently by Maxwell.[9]

It is interesting finally to note the type of plane mirror retinoscope (skiascope) used by Clapp[10] in his extensive investigation concerning the accuracy of retinoscopy in objective refraction. The enviable results recorded were obtained by using an instrument the bulb of which had a long horizontal filament which created an artificial band in the illuminating spot of light. It was necessary to find the point of reversal in both the lesser and the greater curvatures, "the reflection

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6. Elliot, R. H.: Errors of Refraction, Brit. J. Ophth. **2**:313-322, 1918; cited by Copeland.[7]

7. Copeland, J. C.: Personal communication to the author.

8. Copeland Refractoscope Acceptable, report of the Council on Physical Therapy, J. A. M. A. **105**:1982-1983 (Dec. 14) 1935.

9. Maxwell, J. T.: Outline of Ocular Refraction, Omaha, Medical Publishing Company, 1937.

10. Clapp, C. A.: Accuracy of Skiascopy as Based upon a Study of Fifteen Thousand Eyes, Am. J. Ophth. **7**:523-526, 1924.

of the filament being a right angle to the curvature to be determined." I regard this as an additional verification of the principle of the streak retinoscope.

STREAK RETINOSCOPY

The theory underlying streak retinoscopy, as was originally pointed out by Wolff, is precisely the same as that underlying all other types of retinoscopy. The general principles of retinoscopy need not be reviewed here, since excellent accounts are available in the books by Thorington,[11] Duke-Elder,[12] de Schweinitz,[13] Berens [14] and Maxwell.[9] A complete description is given by Pascal.[15]

Two optical systems are involved in retinoscopy, the first of which is the illumination system by which light is directed into the eye to illuminate a portion of the retina. The retina then acts as a secondary source of light for the second optical system, the observation system. The observer is concerned with the fundus reflex, an image of the retinal illumination which appears to lie in the plane of the pupil. The form and behavior of the fundus reflex depend on the type of retinal illumination provided by the retinoscope employed and on the optical construction of the eye as a whole. With the streak retinoscope the fundus reflex appears as a clear, bright band or ribbon of light in the pupil more or less parallel to the streak of light from the instrument. The reflex is brighter and the boundary between light and shadow sharper and more clearcut than with other retinoscopes because the source of illumination lies close to the mirror and because of the focusing lens system employed. The reflex is band shaped because the illuminating streak of light, being narrower than the aperture of the pupil, illuminates a narrow, rectangular area of the retina. Since the fundus reflex is an image of the retinal illumination, it likewise is band shaped. Contrary to the fundus reflex seen with the plane mirror retinoscope, this band-shaped fundus reflex has no relation to the presence or absence of astigmatism. The reflex disappears, and the pupil is filled with light when the mirror of the retinoscope lies at the point of reversal or far point of the lens system of the eye with accommodation at rest.

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11. Thorington, J.: Refraction of the Human Eye, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1930.

12. Duke-Elder, W. S.: The Practice of Refraction, ed. 2, Philadelphia, P. Blakiston's Son & Co., 1935.

13. de Schweinitz, G. E.: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924.

14. Berens, C.: The Eye and Its Diseases, Philadelphia, W. B. Saunders Company, 1936.

15. Pascal, J. I.: Modern Retinoscopy, Including the Principles and Practice of Velonoskiascopy, London, Hatton Press, Ltd., 1930.

Movements of the fundus reflex are observed and interpreted with streak retinoscopy in the ordinary manner, except the light band itself is watched rather than the bordering shadow. With modern streak retinoscopes the angularity of the emergent light and the width of the streak can be varied, or the light rays can be crossed in front of the instrument (concave mirror effect). This adjustment is prone to cause confusion. For simplicity of use, I adjust the streak, light uncrossed, to a width of 4 to 6 mm. at a working distance of 1 meter. No further adjustments are made. With this plano mirror system a "with" movement of the fundus reflex denotes hypermetropia, emmetropia or myopia less than the strength of the lens equivalent to the working distance. An "against" movement denotes myopia of an amount greater

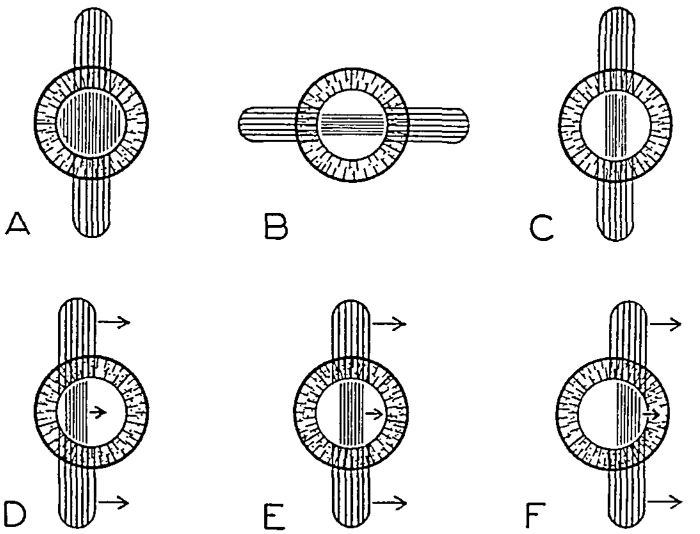

Fig. 1.—*A*, neutral reflex. The pupil of the eye is entirely filled with light. *B* and *C*, the band-shaped fundus reflex in the pupil parallel to the retinoscopic streak. *D*, *E* and *F*, diagrams showing "with" movement, both reflex and streak moving in the same direction.

than the lens equivalent to the working distance. It need only be remembered that light crossing in front of the observer, as in the higher grades of myopia, gives an "against" movement; light emerging from the eye which does not cross in front of the observer gives a "with" movement.

The speed of movement of the fundus reflex also provides useful information concerning the refraction of the eye. The higher the refractive error, either myopic or hyperopic, the slower is the movement of the reflex. At the point of reversal or neutralization of the reflex, when the pupil is either completely dark or completely filled with light, the speed of movement may be considered infinite.

A most important advantage of streak retinoscopy lies in the accuracy with which the axis of astigmatism can be determined. When an astigmatic eye is examined with the streak retinoscope, the fundus reflex exhibits a number of phenomena closely related to those observed by Lindner [16] with the cylinder retinoscope. In a hypothetic case in which at a given working distance the reflex in one meridian has been neutralized, if the streak of light is turned 90 degrees and then passes exactly through the axis of astigmatism, a bright, sharply defined reflex band is seen. When the movement of the reflex is tested, both the reflex and the light streak are seen to move in directions exactly parallel, either with or against. If the streak is rotated a few degrees away from the axis of astigmatism, the reflex becomes much more poorly defined and

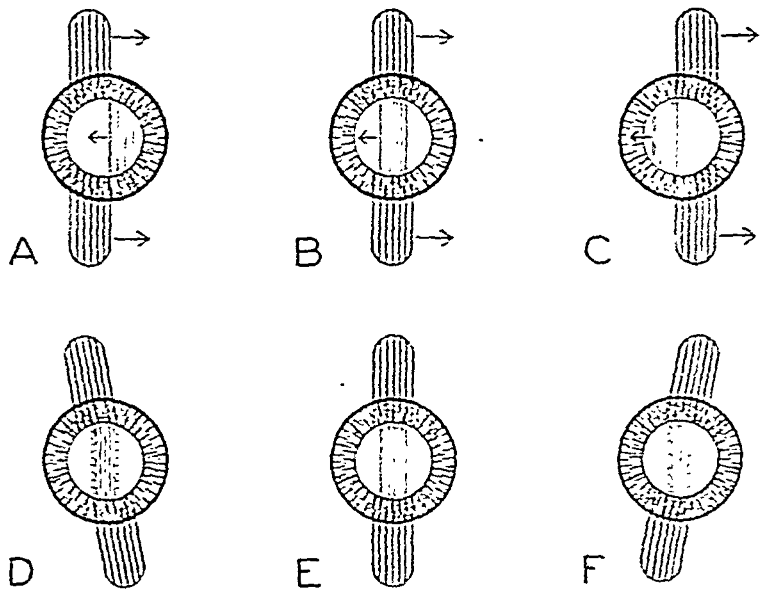

Fig. 2.—*A*, *B* and *C*, diagrams showing "against" movement, streak and reflex moving in opposite directions. *D*, *E* and *F*, diagrams of a 90 degree axis of astigmatism. A bright, clearly defined reflex is seen when the streak passes exactly through the axis of astigmatism as in *E*. Rotation of the streak, *D* and *F*, decreases the clarity of the reflex, but it remains in the astigmatic meridian.

tends to remain fixed in the astigmatic meridian, producing a break in the alinement between the streak of light on the face and the reflex in the pupil. A peculiarity in the movement of the reflex is now noted also. It moves in a direction oblique and rather eccentric to that of the streak, tending to assume a position intermediate between that of the real axis of astigmatism and the streak of light on the face. The exact axis of very low astigmatic errors is easily found by rotating the streak until both streak and reflex move in parallel directions.

16. Lindner, K.: Die Bestimmung des Astigmatismus durch Schattenprobe mit Cylindergläsern, Berlin, S. Karger, 1927.

The magnitude of the astigmatic error can be measured readily by neutralizing with cylinders the reflex in the astigmatic meridian. With the use of cylinders an additional check can be made on the axis of astigmatism. If the cylinder is improperly placed, the following phenomena (Lindner) appear: (1) mixed astigmia, "with" movement in one meridian and "against" movement in the other; (2) oblique astigmia, with the astigmatic reflex oblique to the axis of the cylinder in place, and (3) mobile astigmia, in which the principal optical meridians have apparently shifted.

With the foregoing principles in mind, a procedure suitable for routine ocular refraction with the streak retinoscope is readily designed. When one becomes experienced and expert in the method, subtle differences in the appearance of the reflex enable one to make almost automatic judgments and arrive at accurate retinoscopic estimations with exceeding rapidity. The procedure to be outlined is that which I use:

The patient is seated in the examining chair, the room is darkened, and the phoropter is adjusted. The use of the phoropter here as with other types of retinoscopy is most convenient and saves much time. The patient is directed to focus on a small light 20 feet (6 meters) distant, or with cycloplegia the patient may look into the retinoscopic mirror or at the illuminated disk adjacent to it. The observer sits at a distance of either 66 cm. (26 inches) or 1 meter (40 inches) in a position as close as possible to the visual axis of the eye to be examined. A lens representing the working distance may or may not be placed before the patient's eye at this time, according to the operator's preference. The reflex is somewhat more easily studied if no lens intervenes.

As soon as the streak is thrown on the pupil, a white band of light parallel to the axis of the streak is seen. By an adjustment on the handle of the instrument the streak is rotated about the pupil and the characteristics of the fundus reflex studied. Is the band wide or narrow? Does it move quickly or slowly? Is it clearcut or indistinct? Has it the same characteristics in all meridians? The expert is able to infer much from this initial inspection. As a rule, a narrow band denotes hyperopia or hyperopic astigmatism; a wide band, myopia or myopic astigmatism. When the band is equal in all meridians there is no astigmatism. A "with" movement of the reflex means that the far point of the eye lies beyond the observer; an "against" movement, that it lies between the observer and the patient.

The reflexes are now neutralized systematically. If a "with" movement is observed in all meridians, plus spheres are added until a neutral reflex is obtained, usually first in the meridian in which the reflex is less distinct. If a sphere neutralizes the reflex in all meridians, there is no astigmatism. If a band-shaped reflex having a "with" movement

remains in one meridian, astigmatism is present, and the axis should now be carefully determined as previously outlined. The reflex in the astigmatic meridian is next neutralized with plus cylinders. Finally, the value of both cylindric and spherical corrections is checked by shortening the working distance a few centimeters, whereupon all meridians should show a "with" movement of the reflex, becoming neutral again when the original working distance is resumed. The final retinoscopic measurement is found by adding algebraically to the lenses in place a minus sphere representing the working distance. For 66 cm., a minus 1.5 diopter sphere is added; for 1 meter, a 1 diopter sphere is added. It is seen that this procedure is applicable to all cases of hyperopia and of simple and compound hyperopic astigmatism.

If on initial inspection an "against" movement of the reflex appeared in all meridians, minus spheres are added to neutralize the reflex. If astigmatism is present, a spherical lens will not neutralize all meridians. In this case the strongest meridian is neutralized with minus spheres, giving a neutral reflex in one meridian and a "with" movement in the meridian at right angles to it. The axis of astigmatism is now determined, the astigmatic reflex neutralized with plus cylinders and the value of both lenses checked by the approach-receding method as before. A final correction is máde for the working distance. In the neutralization of "against" movements, since "against" movements are harder to see than "with" movements, the error at first may be overcorrected with minus lenses to produce a "with" movement, following which the strength of the lens is decreased until the reflex is neutralized. The foregoing procedure can be used in cases of myopia and of simple and compound myopic astigmatism.

In mixed astigmatism, showing "with" movement in one meridian and "against" movement in the other, the "against" movement is first neutralized with minus spheres, then the "with" movement is neutralized with plus cylinders.

The subjective test may then be started with the correction for the working distance, as determined retinoscopically, in place.

When one is beginning to use the streak retinoscope it is best to study the reflex in dilated pupils. After experience has been gained, a dilated pupil is no longer necessary, and, indeed, it is often an advantage to do retinoscopic examination on normally contracted pupils, for then only the reflex in the visual zone of the pupil is seen. Another advantage peculiar to the streak retinoscope is that the reflex is narrow enough and sufficiently well defined that retinoscopic examination can be carried out routinely without the use of mydriatic drugs not otherwise indicated.

COMMENT

Accuracy in refraction and how to achieve it is a subject that has been discussed many times both before and since the appearance nearly thirty years ago of Edward Jackson's well known article on the subject.[17] It is now generally accepted that refractive errors may be responsible for widespread symptoms throughout the body and that ametropia even of low or moderate degree should be fully corrected. It is further realized that every inaccuracy in the estimation of ametropia will lessen the chance of relieving the patient. In spite of the confusing array of instruments and methods at present available for measuring refractive errors, much of the refraction done today should probably be classified as "inexpert." Many ophthalmologists have, as medical students or even later, undergone refraction repeatedly before obtaining the relief so easily possible with proper correction. More pertinent evidence in this regard is provided by the large number of patients consulting the ophthalmologist who are already wearing glasses which were or have become grossly inaccurate. If the practical results of refraction are to be improved, it is of the highest importance that methods and procedures most likely to be successful in the hands of those who use them should be developed and emphasized in writing and in instruction.

Accurate refraction requires a careful objective as well as subjective examination. In my opinion there is a widespread tendency to overemphasize numerous refinements of subjective testing while minimizing the value of objective results, with slight appreciation of the intimate, dependent relation that exists between the two. The subjective test is markedly facilitated if one is able, from the retinoscopic findings, to begin the test with approximately the required sphere and cylinder in place, the axis of the latter being properly placed. Under these circumstances one has all the advantages of solving a problem in algebra, knowing the answer in advance. The subjective test then does not last so long as to exhaust the interest and endurance of the patient when the results of the most delicate subjective tests become progressively less accurate and more confusing.

There is, of course, no doubt that ordinary plane mirror retinoscopy has produced results eminently satisfactory in the hands of some operators. The criticism and doubts of others, however, show that success has not been universal. I believe that streak retinoscopy is inherently an easier and more accurate method of retinoscopy, producing results more clearcut and easier to interpret. The method is scientifically grounded, simple in practical application and deserving of a wider trial than has yet been afforded.

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17. Jackson, E.: Accuracy in the Measurement of Refraction, Ann. Ophth. **18**:703-712, 1909.

SUMMARY

The development and use of the streak retinoscope from its invention in 1900 by Hugo Wolff is reviewed. The use of the streak retinoscope is compared with that of the ordinary plane mirror retinoscope, and a procedure suitable for routine streak retinoscopy is outlined. Among the advantages of the method are: the clarity of the fundus reflex, the ability to measure accurately the refraction in different meridians of the eye individually, the accuracy with which the axis of astigmatism can be determined and the ability to perform retinoscopic examination routinely with contracted pupils.

HODGKIN'S DISEASE OF THE LID

REPORT OF A CASE

DANIEL KRAVITZ, M.D.
BROOKLYN

Involvement of any part of the eye with Hodgkin's disease is sufficiently rare to warrant reporting. It becomes increasingly interesting when it occurs in a person of an age far beyond that at which the disease is suspected and rare indeed when the diagnosis is established through the intermediary of the ophthalmologist.

REPORT OF CASE

C. S., a woman aged 82, was seen on Dec. 12, 1937, for refraction. There was sclerosis of both lenses, and the best vision obtained after correction was 20/70 in the right eye and 20/100 in the left. A small cystic tumor was palpated at the left superior orbital border near the inner angle.

On April 20, 1938, the patient returned because of ptosis of the left upper lid. The tumor had increased greatly in size, and as it was felt that it was interfering with elevation of the lid, removal was advised.

On April 25, at the Brooklyn Eye and Ear Hospital, a tumor a little over 1 inch (2.5 cm.) long was removed. It was firm in consistency and was enveloped in a thick white capsule. The tumor was dissected from the underlying periosteum without any difficulty except at its inner end, where it merged with the underlying structures and could not be dissected but had to be cut away. It was felt that the growth was a fibroma.

After operation the lid could be elevated to a considerable extent. In a few weeks the remaining tumor tissue had increased in size, the ptosis had returned, and two similar tumors had appeared along the upper border of the left orbit.

To my great surprise, on June 1 I received a communication from Dr. DeVeer, the pathologist at the hospital, stating that the tumor had all the histologic features of Hodgkin's disease. Reexamination at this time failed to reveal any enlargement of the superficial lymph glands of the neck or face. A roentgenogram of the chest on June 30 did not show any masses or enlarged mediastinal lymph nodes. The blood count was normal except that it showed a mild anemia. Roentgen therapy was advised.

The members of the family remembered that a few years past the patient had been treated with roentgen irradiation for enlargement of the glands on the sides of the face at the Brooklyn Cancer Hospital. Inquiry elicited the following history from this hospital:

"The patient came to the hospital for swelling on each side of the face. A diagnosis of bilateral parotitis was made, and she was treated with roentgen irradiation once weekly from March 21 to May 11, 1934, after which the swelling

Read before the American College of Surgeons at the Brooklyn Eye and Ear Hospital, Oct. 19, 1938.

disappeared. She returned in October 1934, because of a cystlike swelling of the right lower lid. Some small shotty lymph nodes of the superficial cervical glands on the right side were observed. The patient was referred to the ophthalmic clinic and disappeared from observation. No biopsy was made."

The patient left my care and went to the Memorial Hospital, New York, for treatment. The following report was received from this hospital:

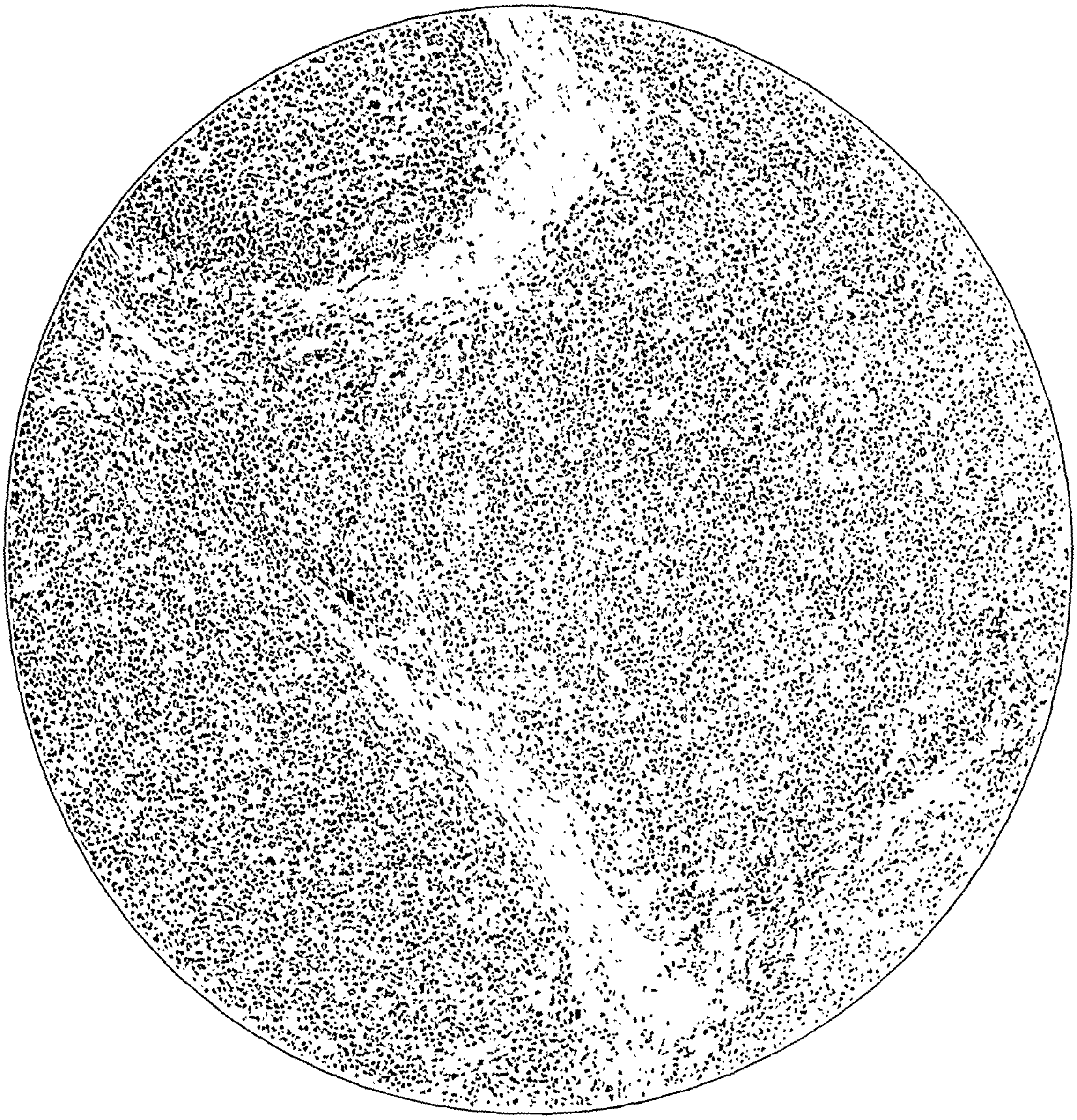

Fig. 1.—The tumor tissue under low magnification, showing considerable pleomorphism. Giant cells and hyperchromatic nuclear masses are numerous in this field. There is also some fibrosis.

"The diagnosis on histologic examination of some aspirated material was dense uniform lymphoid tissue consistent with Mickulicz' disease. The diagnosis based on examination of some material from the tumor sent from the Brooklyn Eye and Ear Hospital was malignant tumor, classified as giant cell reticulum cell lymphosarcoma."

Under roentgen therapy the swelling of the glands on each side of the face disappeared, only to return more markedly in a few weeks. Further roentgen

therapy again caused the swelling to disappear. Two months prior to the writing of this report, signs of pressure in the chest began to develop. A roentgenogram of the chest showed large masses of glands. However, because of the patient's poor physical condition, roentgen therapy of the chest was not deemed advisable. She is still under treatment at the Memorial Hospital.

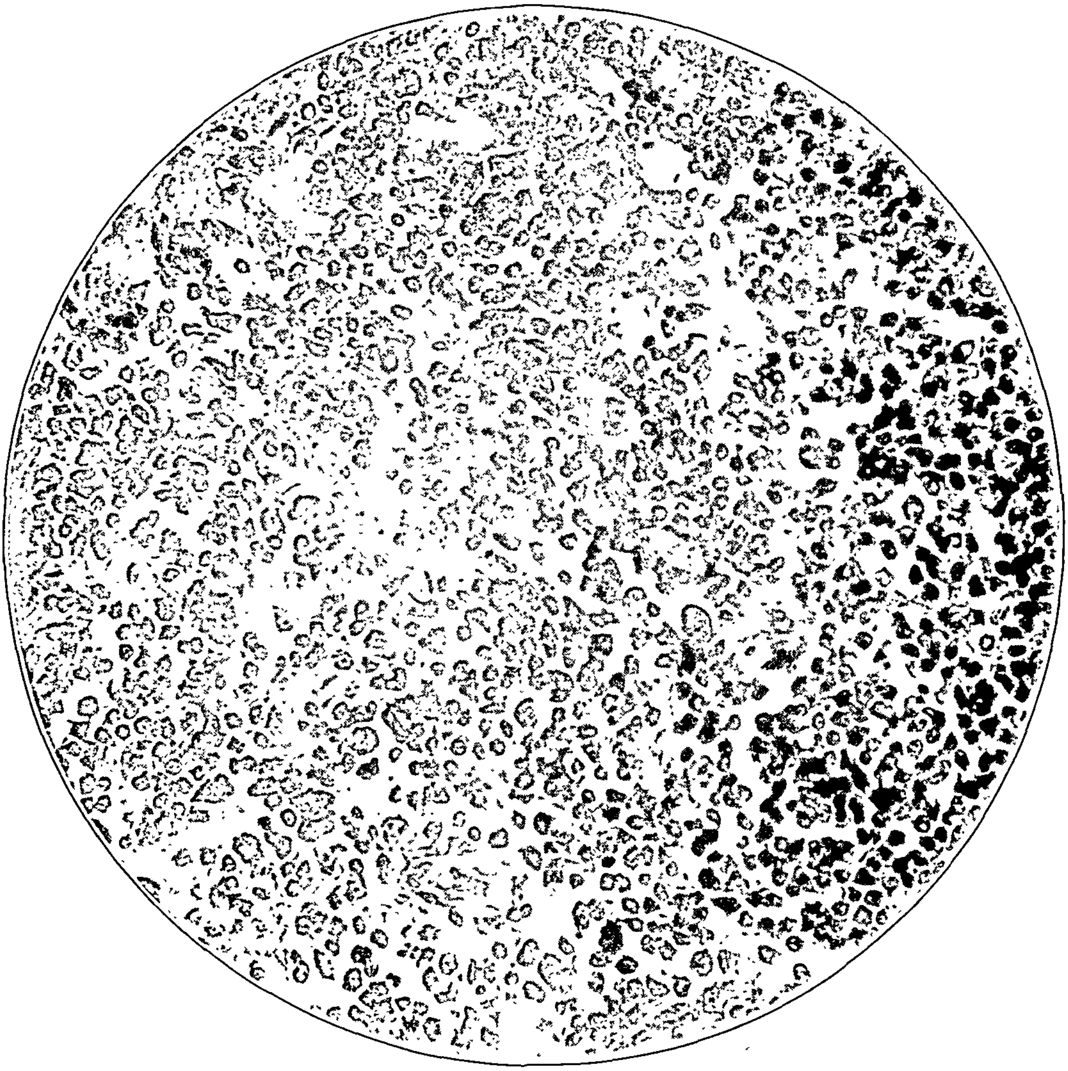

Fig. 2.—A section under higher magnification, showing giant cells of the Reed type, numerous reticulum cells and many leukocytes.

COMMENT

The differences in the diagnosis of different pathologists from histologic examinations of material from the same patient only emphasize the difficulties in classifying this most interesting disease entity.

In 1832, under the title of "Some Morbid Appearances of the Absorbent Glands and Spleen," Hodgkin [1] described 7 cases of a new disease entity. The condition in only 2 of these cases has subsequently been found to fit the description of the disease which now bears his name. The condition in some of the cases was tuberculosis and in the others, leukemia.

Classification of the disease is difficult. Horder [2] stated that he was not certain whether to classify it as a neoblastoma or as an inflammation. The disease shares the features of each condition. Its main expression is tumor formation, but the existence of fever, the occurrence of natural remissions and the histologic characteristics of the many lesions are identical with the course the same tissue takes when subjected to microbic infection. Boyd [3] also expressed the belief that in spite of the fatal outcome the pleomorphism shown by the histologic picture is one usually associated with a chronic inflammatory lesion rather than with a malignant process. According to him, this disease may occupy an important borderline position between inflammation and neoplasia. The disease is frequently preceded by a sore throat, and in the majority of cases the cervical glands are first involved. This occurrence caused Horder [2] to express the opinion that the infective agent enters in the vicinity of the cervical glands. He also stated that an infection of known nature, such as a tuberculous or a pyogenic infection, may precede the pathologic agent at its point of entrance.

The exact causation of the disease is not known. Probably the most persistent and insistent view has been that it is some form of tuberculosis. Sternberg [4] maintained that it is due to the toxin of tuberculosis. Ewing [5] demonstrated that tuberculosis does not develop in guinea pigs inoculated with material from patients with lymphadenoma until from nine months to a year afterward. Gordon [6] was unable to cultivate the

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1. Hodgkin, T.: Some Morbid Appearances of the Absorbent Glands and Spleen, Med.-Chir. Tr. **17**:68, 1832.

2. Horder, T.: A Clinical Concept of Lymphadenoma or Hodgkin's Disease, in Horder, T., and others: Rose Research on Lymphadenoma, Bristol, England, John Wright & Sons, Ltd., 1932.

3. Boyd, W.: Text Book of Pathology, ed. 3, Philadelphia, Lea & Febiger, 1938, p. 319.

4. Sternberg, C.: Ueber eine eigenartige unter dem Bilde der Pseudoleukämie verlaufende Tuberkulose des lymphatischen Apparates, Ztschr. f. Heilk. **19**:21, 1898.

5. Ewing, J.: Neoplastic Diseases, ed. 3, Philadelphia, W. B. Saunders Company, 1928, p. 407.

6. Gordon, M. H.: Studies of the Etiology of Lymphadenoma, in Horder, T., and others: Rose Research on Lymphadenoma, Bristol, England, John Wright & Sons, Ltd., 1932.

tubercle bacillus from material from patients with typical Hodgkin's disease. He expressed the belief that too much attention has been paid to tuberculosis as a cause, particularly since it has been shown that lesions resembling tuberculosis microscopically and macroscopically may be produced by certain fungi, yeasts and molds.[7] Gordon also failed to substantiate the claim that Hodgkin's disease is a form of mycosis.[8] Fränkel and Much [9] found gram-positive, nonacid-fast bacillary and granular bodies which they thought might be the cause of Hodgkin's disease. This finding was confirmed by Kusunoki.[10] A diphtheroid bacillus was found by Bunting and Yates.[11] The latter were able to produce granular swellings which histologically resembled the granuloma of Hodgkin's disease. Rosenow confirmed their claims, and he and Billings [12] treated a number of patients with a vaccine prepared from this organism, with favorable results. Pröscher and White [13] claimed that the disease is due to a spirochete. However, none of the foregoing claims were substantiated by the research work of Gordon, who expressed the belief that the most recent evidence in favor of a filtrable virus as its cause is the most promising.

Gordon [14] injected some emulsified gland from patients with Hodgkin's disease into the brains of rabbits and guinea pigs. Symptoms of marked involvement of the brain and spinal cord, such as those of paraplegia, appeared. Postmorten examination showed signs of severe inflammation of the nerve tissues. Lymphocytic infiltrations were not visible. Allen and Mercer [15] reported 2 cases in which clinical and

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7. (*a*) Schenck, B. R.: On Refractory Subcutaneous Abscess Caused by a Fungus Possibly Related to the Sporotricha, Bull. Johns Hopkins Hosp. **9**:286, 1898. (*b*) Stollard, J. L., and Cutler, E. C.: Torula Infection in Man, Monograph 6, Rockefeller Institute for Medical Research, 1916.

8. Dias, E. C.: Adenomycosis, New Orleans M. & S. J. **70**:598, 1918.

9. Fränkel, E., and Much, H.: (*a*) Bemerkungen zur Aetiologie der Hodgkinschen Krankheit und der Leukämia lymphatica, München. med. Wchnschr. **67**: 685, 1910; (*b*) Weitere Untersuchungen über Lymphogranulomatose, Ztschr. f Hyg. u. Infektionskr. **9**:39, 1923.

10. Kusunoki, M.: Zur Aetiologie der Lymphomatosis granulomatosa, Virchows Arch. f. path. Anat. **214**:184, 1914.

11. Bunting, C. H., and Yates, J. L.: Cultural Results in Hodgkin's Disease, Arch. Int. Med. **12**:236 (Aug.) 1913.

12. Billings, F., and Rosenow, E. C.: The Etiology and Vaccine Treatment of Hodgkin's Disease, J. A. M. A. **61**:2122 (Dec. 13) 1913.

13. Pröscher, F., and White, W. C.: Ueber das Vorkommen von Spirochetes bei pseudoleukämischer Lymphdrüsenhyperplasie, München. med. Wchnschr. **54**: 1868, 1907.

14. Gordon, M. H.: Recent Advances in the Pathology and Treatment of Lymphadenoma, Proc. Roy. Soc. Med. **27**:1035, 1934.

15. Allen, I. M., and Mercer, J. O.: Spinal Symptoms with Lymphadenoma, J. Neurol. & Psychopath. **17**:1, 1936.

autopsy observations matched the foregoing experimental findings. The spinal fluid in each case gave evidence of an inflammatory reaction. These authors agreed with Gordon that this is evidence in favor of a filtrable virus as the cause of the disease.

McJunkin [16] called attention to the resemblance of certain cytologic changes produced by the Rous virus in experimental fowl sarcoma to those present in lymphadenoma. Coley [17] maintained that the clinical features of Hodgkin's disease so closely resemble those of sarcoma that it is often impossible to differentiate the two conditions, and even the pathologists are divided in their opinion when looking at a specimen. Coley expressed the belief that this fact furnishes additional evidence in favor of the infectious origin of sarcoma. Ewing [5] has shown that Hodgkin's disease may become sarcomatous—Hodgkin's sarcoma. However, he stated that the sarcoma is the result of chronic irritation on the neighboring endothelial cells or that as a result of vigorous proliferation of the lymphoid cells these cells become sarcomatous. Turnbull,[18] however, called the disease a lymphosarcomatoid condition and regarded it as an inflammatory reaction.

As previously stated, the cervical glands are the most frequently affected; this is thought to be the result of a previous local irritation in the mouth and tonsils. The glands are hard and discrete, and the glandular swelling then spreads to the other parts of the body. Later the glands may fuse, as in tuberculosis. Rarely the parotid glands may be first involved, and the disease may resemble Mikulicz' disease. Symers [19] and Ewing,[19a] however, stated that the superficial glands are not first involved but that they are the outward manifestation of an internal disorder. According to them, the thoracic and abdominal glands are first involved. Osler [20] called attention to a rare type of disorder called lymphoid ossium in which there are multiple tumors of the bone marrow and the periosteum. He was doubtful about considering this condition Hodgkin's disease. However, in Allen and Mercer's [15] cases, definite osseous and periosteal invasions were found.

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16. McJunkin, F. A.: Histologic Resemblance of Rous Chicken Sarcoma No. 1 to Hodgkin's Granuloma, J. Cancer Research **12**:47, 1928.

17. Coley, W. B.: (*a*) Further Evidence in Support of the Theory That Hodgkin's Disease Is a Type of Sarcoma, Tr. Am. S. A. **26**:560, 1908; (*b*) A Report of Recent Cases of Inoperable Sarcoma Successfully Treated with Mixed Toxins of Erysipelas and Bacillus Prodigiosus, Surg., Gynec. & Obst. **13**:174, 1911.

18. Turnbull, H. M., cited by Rolleston, H.: Lymphadenoma (Hodgkin's Lymphogranuloma), Lancet **2**:1209, 1925.

19. Symers, D.: Clinical Significance of Pathological Changes in Hodgkin's Disease, Am. J. M. Sc. **167**:157, 1924.

19a. Ewing, cited by Rolleston,[26] p. 1210.

20. Osler, W.: The Principles and Practice of Medicine, ed. 8, New York, D. Appleton and Company, 1912, p. 749.

Histologically, the disease is characterized by marked variation in the picture and a pleomorphism. One sees mainly large pale cells of an "epithelioid" type, so that there is a resemblance to the hyperplastic form of tuberculosis. These cells are derived from the reticuloendothelial cells. More characteristic is the presence of large reticulum cells, giant cells, which are seen in the process of division. Many are multinuclear, and some are mononuclear; when they are single, they are often convoluted or ring shaped. The nuclei are large and stain darkly against a faintly staining cytoplasm. These are the Sternberg, or Dorothy Reed, cells. The presence of these cells is essential to the diagnosis of lymphadenosis. Eosinophilic leukocytes in great profusion complete a characteristic picture. These may be absent. However, all pathologists agree that when they are present a diagnosis of Hodgkin's disease is favored.

Lymphocytes, plasma cells and polymorphonuclear cells may all be present to complicate the picture. The last are usually secondary to necrosis. An increase in the fibrous tissue is another change, in the absence of which no lesion is considered quite typical. This fibrosis increases with time and may become dense. At times the microscopic picture may be more neoplastic in appearance, with many mitotic figures and a tendency to invasion of the surrounding tissue. This is the picture presented by the Hodgkin's sarcoma of Ewing.

Hodgkin's disease is the result of an irritation of the reticuloendothelial tissue. It is interesting to note that the study of the reticuloendothelial system originated with the eye. In 1863 von Recklinghausen [21] described ameboid cells in an inflamed cornea and in the omentum of different animals. These cells could be distinguished from pus corpuscles. He expressed the belief that they came from the fixed connective tissue cells. Further study and elaboration of this complex system were carried on by the process of vital staining.

Maximow [22] objected to the inclusion of the term endothelial in the picture. According to him, the reticulum is not composed of well defined units but of a series of spongelike syncytia of nuclei and protoplasm. This reticulum has multipotentialities which are retained in adult life, just like the mesoderm from which it is derived. The reticulum is distributed as a ground substance in the lymphoid and myeloid tissue and in the red pulp in the spleen. In the lymphatic glands it lines the lymph sinuses. Under the proper stimuli, individual cells separate and give origin to lymphocytes, histiocytes, free macrophages and fiber-bear-

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21. von Recklinghausen, F.: Ueber Eiter und Bindegewebskörperchen, Virchows Arch. f. path. Anat. **28**:157, 1863.

22. Maximow, A., in von Möllendorff, W.: Handbuch der mikroskopischen Anatomie des Menschen, Berlin, Julius Springer, 1927, vol. 11, pt. 1.

ing cells of various kinds. Lang [23] has furthermore proved the possibility of direct myeloid transformation. Maximow stated that reticulum is derived from the mesoderm and not from the endothelium of the vascular and lymph channels; therefore reticuloendothelium is a misnomer, because it links two different types of cells. Medlar [24] considered the Dorothy Reed cells to be megakaryocytes derived from the bone marrow.

The differential diagnosis is still beset with many difficulties, and, as Watson [25] stated, a differentiation between Hodgkin's lymphosarcoma and leukemia may be impossible from the study of one node.

The close association between Hodgkin's disease and tuberculosis has been noted from the beginning. The giant cells in the two conditions are distinct. The glands in Hodgkin's disease have a tendency to remain discrete, and the steady, progressive and fatal termination is different from the clinical course of tuberculosis. Rolleston [26] explained the close association of the two conditions on the basis that Hodgkin's disease either activates a tuberculous process or else makes the glands more predisposed to a tuberculous infection.

Hodgkin's disease is differentiated from leukemia by the almost normal blood count, whereas in leukemia there is a high lymphocyte count. The differentiation from the aleukemic form of leukemia is more difficult, but histologic sections do not show the giant cells and the eosinophils of Hodgkin's disease.

The greatest difficulty lies in differentiating Hodgkin's disease from lymphosarcoma, which may resemble it clinically. In lymphosarcoma the glands form larger masses. This condition is more frequent in the fourth decade of life, while Hodgkin's disease occurs more frequently in the second and third decades. The consistency of the tumor in lymphosarcoma is not as hard as that in Hodgkin's disease. Histologically, the predominant cell is the lymphocyte. The presence of eosinophils and the absence of a tendency to invasion and infiltration of the surrounding tissue point to Hodgkin's disease. While all the authors stated that the two diseases are distinct entities, they expressed the opinion that all diseases of the blood may have a common etiology. Different glands in the same patient may show the characteristics of each of the diseases. Particularly difficult to diagnose are the early stages

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23. Lang, F. J.: Experimentelle Untersuchungen über die Histogenese der extramedullären Myelopoese, Ztschr. f. mikr.-anat. Forsch. **4**:417, 1926; Myeloid Metaplasia, Folia haemat. **43**:95, 1930.

24. Boyd,[3] p. 831.

25. Watson, C. J., in Downey, H.: A Handbook of Hematology, New York, Paul B. Hoeber, Inc., 1938, vol. 4, p. 3074.

26. Rolleston, H.: Lymphadenoma (Hodgkin's Lymphogranuloma), Lancet **2**:1209, 1925.

when transitional forms are present. This caused Watson [25] to state that there may be a fundamental relation between all diseases of the blood.

The course of Hodgkin's disease is progressive; the anemia increases, and there are episodes of fever, which may last for days, the Pel-Ebstein type of fever. The glands enlarge, so that there may be pressure symptoms in the chest or abdomen. The skin becomes bronzed, as in Addison's disease. Itching is a marked and fairly constant symptom. Gradually the patient dies of inanition or most probably from an intercurrent infection.

Treatment is palliative and prolongs the remissions. Arsenical preparations have proved of definite benefit. The best treatment is roentgen irradiation of the enlarged glands. It is remarkable to see the swelling disappear. This form of therapy is of greatest benefit when the disease is new. Later, when fibrosis is marked, the results are poor. However, eventually the swelling of the glands returns again and again, and a fatal termination is inevitable. It had been noticed that enormous lymphomatous masses may disappear subsequent to an intercurrent disease, such as erysipelas. On this basis an unfiltered mixture of erysipelas and prodigiosus cultures (Coley's) has been injected with good results, probably because of the induced fever.

The duration of the disease is variable. Rarely there may be an acute termination. In the average case the condition lasts from one to two years. When the disease is chronic, it may last many years.

Dr. Arnold DeVeer prepared the pathologic material and chose the most representative parts for photography.

NEW INSTRUMENTS FOR OCULAR OPERATIONS

EDWARD RICHARD GOOKIN, M.D., WASHINGTON, D. C.

Description of three instruments which I have completed recently for use at operations on the eye are presented.

The first of these, the clamp and squint hooks, consists of three instruments in one. The squint hooks are used individually but may be

A, the clamp and squint hooks. *B*, the two instruments combined to make the muscle clamp. *C*, the needle guide. *D*, the improved spatula.

combined to make the muscle clamp. The latter makes any operation on a muscle much more simple, and the cost is less than that of instruments previously used.

The second instrument is a needle guide for the purpose of preventing any laceration of tissue during the process of suturing. The guide is pressed against the tissue by an assistant, and the needle and suture are drawn through the opening after the plan utilized in the modern sewing machine.

I also find that the guide is handy in suturing during plastic work on the skin.

The third instrument is an improved spatula, which I find better than other instruments for replacing an iris following extraction of cataract, iridectomy and other operations. I have also used it frequently for raising a pterygium from tissue below; with the spatula held in one hand, the operation can be completed with scissors held in the other hand.

These instruments are manufactured by E. B. Meyrowitz Surgical Instruments Co., Inc., New York.

ARACHNODACTYLY

Report of a Case

TELFORD I. MOORE, M.D., SPOKANE, WASH.

The case of arachnodactyly reported here is interesting because the condition occurs singly in a perfectly normal family (3 siblings) and because of the train of events leading to the diagnosis.

Photographs of the patient.

J. B., aged 6 years, was brought in for examination as required for admission to the state school for the blind. He had been examined at the age of 4 by an oculist (now deceased), who after several attempts to improve the extremely poor vision gave the mother a discouraging prognosis, and nothing was done.

External examination of the eye showed that the patient's far point grossly was about 3 inches (7.6 cm.). The lids, the position of the eyes, conjunctiva, the lacrimal apparatus and the tension on palpation were normal. The pupils were small and irregular. They reacted sluggishly to light and in accommodation. The irides were both tremulous. It was impossible to dilate the pupils more than 3 mm. with any of the common mydriatics, including benzedrine.

Slit lamp examination showed the corneas to be clear. The anterior chambers had normal depth and negative aqueous rays. The irides showed normal pigmentation, but there was definite atrophy of the dilator fibers. The lenses could be seen only in the pupillary area and appeared clear.

Ophthalmoscopically, only a good red reflex was obtained. No details could be elicited, even with the subsequent refraction.

Retinoscopic examination was most difficult; at 0.2 meter about a —38.00 sph. was suggested for each eye. This correction was tried for two weeks with the most dramatic change in the patient. At the time of writing he is in the first grade of the public school and is doing good work. Subsequent tests after he learned a few letters revealed vision to be about 20/100 in each eye with the foregoing correction.

COMMENT

Since the most comprehensive article of Burch [1] in 1936 there has been little added to the bibliography of this interesting condition. The accompanying photographs of the patient aid in establishing the diagnosis. The ocular findings fall in line with those previously reported. The usual finding of congenital heart disease was present in this case. The present plans call for a discission and linear extraction of both lenses.

1. Burch, F.: Association of Ectopia Lentis with Arachnodactyly, Arch. Ophth. **15**:645-679 (April) 1936.

Ophthalmologic Reviews

Edited by Dr. Francis Heed Adler

UNILATERAL PAPILLEDEMA

ITS SIGNIFICANCE AND PATHOLOGIC PHYSIOLOGY

E. GERARD SMITH, M.D., M.Sc. (Med.)
LANCASTER, PA.

Although much of the ophthalmic literature deals with papilledema, its unilateral occurrence is mentioned only in widely scattered papers. Unilateral involvement is relatively rare but is nevertheless important and presents a difficult diagnostic problem. When the condition is bilateral certain causes are almost automatically excluded. Many additional factors which complicate the diagnosis must be taken into consideration when the condition is limited to one eye. The term choked disk is assiduously avoided in this paper, because it is often taken to include papillitis or optic neuritis, an entity having a different causation, different pathologic characteristics and symptoms and, to a lesser extent, a different appearance and different sequelae. In their early stages the differentiation of the two conditions may be difficult or impossible. Since a clear understanding of the two conditions is important, for reasons which will be given later, a brief summary of their chief characteristics will be presented here.

In papilledema there is considerable elevation of the nerve head; the arteries may be smaller than normal and the veins enlarged, with great disproportion between the two. There are usually hemorrhages in the retina. Vision may be normal over a long period. The nerve fibers remain clear longer, and the physiologic cup usually persists until the condition is well advanced. Histologically, there is an infiltration which is at first a simple mechanical edema, but if the condition continues, more and more cellular infiltration occurs. The most frequent cause of papilledema is increased intracranial pressure. In optic neuritis, on the other hand, there usually is little if any elevation of the disk.

From the Department of Ophthalmology of the Hospital of the University of Pennsylvania.

Thesis submitted to the faculty of the Graduate School of Medicine of the University of Pennsylvania in partial fulfilment of the requirements for the degree of Master of Medical Science (M.Sc. [Med.]) for graduate work in ophthalmology.

The arteries show little change, and the veins are broad and tortuous. Hemorrhages in the retina are not often present. The vision is usually much impaired from the beginning. The nerve fibers become opaque much more rapidly, and the optic cup is soon obliterated by an exudate. The visual field often shows a central scotoma, while in papilledema there are usually only an enlarged blindspot and slight concentric contraction. Histologically, papillitis is characterized by hyperemia, which accounts for the marked redness of the disk. There is some swelling of the tissues due to an exudate, which is usually serous and plastic or fibrinous and purulent. The latter may change the color to a dull grayish or grayish red tint. Among the more common causes are: general and focal infections, metabolic diseases, toxemias and poisoning (Troncoso). There are, of course, other differential points, and those cited previously could readily be amplified, but this has not been done for the sake of brevity. There are many theories as to the pathogenesis of papilledema. These are of no concern here except in so far as they may throw some light on the factors which limit the papilledema to one eye.

The causes of unilateral papilledema may be classified as follows: ocular and extraocular. The latter may be further subdivided into: (*a*) orbital, (*b*) intracranial and (*c*) general systemic causes. Cases in which one eye is incapable of being affected by papilledema will be discussed under pathologic physiology.

OCULAR CAUSES

Ocular causes include those etiologic factors which are located in the globe or act directly on the globe. The most important factor in this group is reduced intraocular tension. Papilledema due to decreased ocular tension has been observed clinically many times. Elschnig reported a case of keratomalacia with perforation of the cornea in 1902. He described the microscopic appearance of the disk as "identical to that of papilledema due to brain tumor." Similar cases in which papilledema followed perforation of one sort or another, and histologic confirmation was made, have been reported by Stock in 1903, by Fuchs in 1904 and 1928, by Kampherstein in 1904, by Gilbert in 1910, by Behr and by Inouye in 1912 and by others. A close analysis of these cases does not convince one, however, that inflammation might not have been present. The degree of papilledema in such cases may be high. Schieck reported a case in which as a result of a perforating wound a small fistula remained at the limbus. There were no inflammatory signs of any kind. The disk showed an elevation of 4 D. After closure of the fistula by a plastic operation, with resultant increase in the intraocular tension, the swelling disappeared in several weeks. Scardapane

reported 2 cases of perforation of the cornea with subsequent papilledema. In these cases the spinal fluid pressure was elevated. He attributed this elevation to an overproduction of spinal fluid by choroid plexuses irritated by toxins passing from the eye along the optic nerve to the cranial cavity. There is little to substantiate such a theory. Perforation need not necessarily take place in the anterior segment of the eye. Knapp reported a case of retinal detachment in which a Gonin operation was done, which was followed by a swelling of the nerve head amounting to 2 D. In discussing Knapp's paper, Vogt mentioned his own repeated observation of this complication. External trauma without perforation may also bring about a sufficient decrease in the intraocular tension. Polanyi recorded a case of unilateral edema of the nerve head following a nonpenetrating blow. The eye was soft, and the disk was elevated 2 D. On the eighteenth day there was no trace of swelling, and the intraocular tension was 40 mm. of mercury. Polanyi expressed the belief that the hypotension was caused by a disturbance of the ciliary epithelium and that the subsequent hypertension was due to a later irritative reaction with resultant increased secretion. Eppenstein reported a case of glaucoma in which vibratory massage was the only treatment. Seven days after treatment was initiated there were hypotension and a swelling of 2 D., which increased somewhat, then disappeared seven days later without treatment, owing to a recurrent elevation of intraocular tension. Eppenstein expressed the belief that the edema was due to the pressure exerted by the normal tension of intracranial fluid. Carle described a similar case in which papilledema resulted after a sudden reduction in tension due to the use of miotics. Several cases of lowered intraocular tension with papilledema due to phthisis bulbi have been reported by van den Borg. The condition in 1 case followed an attack of measles and in the other tuberculous iridocyclitis and scleritis. In both cases a diagnosis of true papilledema was made on the basis of the histologic studies, but one is justified in questioning the accuracy of the observations.

Papilledema has been produced experimentally by a reduction in the intraocular tension by trephining. Gilbert produced papilledema in a series of rabbits, and Kyrieleis repeated the work with a series of dogs and another series of rhesus monkeys. In these a true papilledema resulted in the absence of any inflammatory reactions, and the author made a thorough histologic study of the specimens. He expressed the belief, however, that papilledema produced in this manner is not like that seen in increased intracranial pressure. He also was of the opinion that it is not the typical sharply marginated "button" or "mushroom" type. The fact that the work was done with animals may account for this.

The only intraocular cause so far described here is reduced intraocular tension. This may be due to perforation. In these cases papillitis cannot be positively ruled out, because infection may have been present. Those cases in which the causative factor was nonperforating trauma or medication strongly suggest that papilledema can be due to lowered intraocular pressure.

CASE 1 (reported by permission of Dr. W. E. Fry).—N. H., a man aged 49, was first seen in February 1935. At that time the tension (Schiötz) was 33.5 mm. of mercury in the right eye and 51.5 mm. in the left eye. The visual fields were characteristic of glaucoma. Examination of the fundi showed in the right eye slight indentation of the disk at the temporal border and in the left eye deep cupping of the disk, typical of glaucoma. Vision in the right eye was 6/5; that in the left eye, with correction, was limited to perception of hand movements. A diagnosis of bilateral glaucoma was made. The patient was treated medicinally until August 1935, at which time an Elliot trephine operation was done on both eyes. On September 5 the tension (Schiötz) in the right eye was 7 mm. of mercury and in the left eye 8 mm. Vision in the right eye was 5/7.5; that in the left eye was 3/100 with correction. On October 17 the vision in each eye was the same as on September 5. Examination of the fundi showed an oval disk and no cupping in the right eye and an atrophic disk and well marked cupping in the left eye. On December 24 vision in the right eye was 5/4; that in the left eye was 1/100 with correction. The tension (Schiötz) in each eye was 7 mm. of mercury. The patient was not seen again until Jan. 27, 1938, at which time vision in the right eye was 6/9; that in the left eye, with correction, was limited to perception of hand movements. Examination of the fundus of the right eye showed the margins of the disk to be hazy. The disk appeared edematous but not elevated. The fundus of the left eye appeared as before. On March 11 vision in the right eye was 6/12; that in the left eye, with correction, was limited to perception of hand movements. Examination of the fundus of the right eye showed elevation of the disk of from 1.5 to 2 D. The edema extended out from the disk to the nasal margin of the macula. There was no change in the fundus of the left eye. Tension on palpation was very low in each eye. On March 21 vision in the right eye was 6/12 with correction. There was no change in the vision in the left eye. The fundus of the right eye showed elevation of the disk of 1 D. Tension was still extremely low.

The patient had no signs or symptoms of hypertension. There were no signs of inflammation, and although the elevation was relatively small, there was definite edema of the nerve head and surrounding tissue. The papilledema was probably due to the lowered intraocular tension. No papilledema developed in the other eye because the nerve was atrophic. As Marchesani pointed out, the glial connective tissue present in atrophic nerves prevents papilledema.

EXTRAOCULAR CAUSES

(*a*) *Orbital Causes.*—The most important orbital causes of unilateral papilledema are neoplasms of any of the structures comprising or contained in the orbit. Such new growths may be of any sort, and

the literature contains reports of various types, including sarcoma of the orbital bones (Rumjantzewa), osteofibroma (van der Hoeve), myeloma (Tahano), cavernous angioma, endothelioma, melanoma (Ewing) and many others. Pfingst reported a case of unilateral papilledema with exophthalmos due to an aneurysm of the ophthalmic artery, and Teulières described 2 cases due to ecchinococcus cysts. Heine studied a case of Mikulicz' disease in which bilateral exophthalmos and unilateral edema of the nerve head were present. An orbital phlegmon may bring about the same result (Kljacko). Tumor of the optic nerve is another cause of unilateral papilledema (Heuss). Grafowa and Mikulinska described a case in which the lower half of the disk was edematous and elevated 3 D., while the upper half was atrophic and flat. Histologic examination showed a partial infiltration of the nerve by a myofibrosarcoma, resulting in atrophy of one half of the nerve fibers due to pressure from thickened septums. The other half of the nerve was distended by lymph in the intercellular spaces. Sinusitis is included in this group of causes. In empyema of a sinus the walls may bulge into the orbit or may cause inflammation of adjacent structures. Belgeri and Arana reported a case of unilateral edema of the nerve head due to sinusitis. When drainage was reestablished by the use of a spray containing a solution of cocaine hydrochloride and epinephrine hydrochloride there was immediate regression of the edema, with sudden restoration of normal vision. Unilateral papilledema in a case summarized by Worms and Chams was attributed to syphilitic involvement of the periorbita and bones. In cases such as the last two papillitis must be carefully excluded.

Case 2 (service of Dr. T. B. Holloway).—R. S., a woman aged 26, was admitted to the hospital on March 12, 1933. The chief complaint was blurred vision in the left eye. Ten days before admission the patient awoke one morning with severe pain in the region of the left eye and the forehead. Vision in the left eye was blurred, the blurring being most marked in the lower temporal field. There was a past history of frequent colds, sinusitis and sore throat. The tonsils and adenoids had been removed ten years previously. Physical examination gave negative results except for thickening of the left middle and inferior turbinates due to pus in the left sphenoethmoid region. Roentgen examination showed bilateral ethmoiditis. There was a slight increase in the density of both antrums. The optic foramens were normal. Ophthalmic examination showed vision of 6/5—2 in each eye. External examination gave negative results. The fundus of the right eye was normal. The fundus of the left eye showed papilledema of 3 D. On March 20 the visual field of the right eye was normal. The field of the left eye showed slight concentric contraction and an enlarged blindspot. The picture was suggestive of a defect in the left inferior nasal quadrant. Ophthalmoscopic examination of the right eye gave negative results. There were many opacities of the vitreous of varying size in the left eye, with numerous areas of exudates and diffuse hemorrhages. The papilledema was slightly increased. On March 21 the visual field of the right eye was normal. The concentric con-

traction in the left eye was more marked, and the quadrant defect was increasing in size. Ophthalmoscopic examination showed no change. On March 24 spheno-ethmoidectomy on the left side was followed by immediate improvement and total regression of the papilledema in six days.

The papilledema in this case was undoubtedly due to ethmoiditis. At operation a considerable quantity of pus was evacuated. Inflamed ethmoid cells may cause pressure on the optic nerve, either directly or indirectly by inflammation or edema of adjacent structures.

Case 3 (reported by permission of Drs. Francis Heed Adler and Francis C. Grant).—G. D. S., a girl aged 12 years, was admitted to the hospital on June 11, 1937. The chief complaint was pain in the left orbital region of three or four months' duration, beginning occasionally in the morning. There was a gradual increase in severity and frequency, the pain never lasting more than a few minutes, with intervals of freedom. Vision in the left eye had gradually been blurring for one month before the patient's admission. Physical examination showed a few tags of tonsil tissue remaining. Many glands were palpable in the neck. Ophthalmic examination on June 8 showed vision of 6/6 in the right eye and 6/9 in the left eye. The visual field of the right eye showed concentric contraction and an elongated blindspot. The visual field of the left eye showed concentric contraction, which was marked for red. The field for red cut to the midline on the temporal side. The blindspot was enlarged. The fundus of the right eye was normal; that of the left eye showed papilledema of 4 D. and a few hemorrhages. External examination did not reveal any exophthalmos. The left corneal reflex was slightly less than the right. Roentgen examination of the skull gave negative results. On June 15 ophthalmic examination showed the right eye to be normal. There were papilledema of 2 D. and a few hemorrhages in the left eye. The visual fields were similar to those seen at the previous examination. An encephalogram made on June 17 was normal. On June 29 ophthalmic examination gave entirely negative results. Vision was 6/6 in the right eye and 6/6—1 in the left eye.

In view of the fairly good vision and the characteristics of the changes in the visual fields and the fundus, a diagnosis of papilledema was made. This case is included in this group because the enlarged glands in the neck were suggestive of a focus of infection in the sinuses, although at that time no definite evidence of sinus disease was found. Furthermore, the papilledema subsided without treatment.

Case 4 (services of Drs. J. C. Gittings and Francis C. Grant).—M. G., a girl aged 11 years, was admitted to the hospital on June 21, 1937. The chief complaint was fainting spells since September 1936, followed by frontal headaches and precordial pain. Weekly attacks occurred up to March 1937. There were no attacks until May 1937, then they increased in frequency. Physical examination showed slight thickening of the mucous membrane in the region of the maxillary and ethmoid sinuses. There was partial obstruction of the left nasal passage. Enlarged glands were palpated in the anterior and posterior triangles of the neck. Ophthalmic examination showed vision of 6/9 in each eye. The fundus of the right eye was normal. Examination of the fundus of the left eye revealed papilledema of 4 D. The left pupil was slightly smaller than the

right. The visual field of the right eye was normal; that of the left eye showed slight concentric contraction and an enlarged blindspot. The patient was removed from the hospital by her parents on June 23. She was readmitted on July 22 because of recurrent fainting spells and convulsive seizures. Physical examination gave the same results as on the first admission. Roentgen examination of the skull gave negative results. An encephalogram made on July 23 was normal. Ophthalmic examination on July 26 showed vision of 6/9 in the right eye and 6/12 in the left eye. The fundus of the right eye was normal. The papilledema in the left eye had subsided to 1 D.

In this case a diagnosis of papilledema was made because of the characteristics of the visual fields, the fundus pictures and the relatively small impairment of vision. Nevertheless, papillitis cannot be definitely excluded. The etiologic factor here was probably the same as that in the preceding cases. The convulsive seizures were thought to be of psychic origin.

(*b*) *Intracranial Causes.*—The most common intracranial cause of unilateral papilledema is tumor. A tumor associated with edema of one nerve head may be located anywhere. The literature includes reports of cases of frontal tumor (Ballado and Adrogué, Foster Kennedy, Haitz, Herrmann, Marchesani, Mylius, Schmelzer, Thies and many others), of chiasmal tumor (Sørensen, Christiansen); of tumor of the sphenoid ridge (Alpers and Groff), of tumor of the cerebellopontile angle (Brückner, Hagens, van der Hoeve and others), of cerebellar tumor (Herrmann) and of tumor of the sella turcica (Vincent). Many other tumors in various locations, too numerous to mention, are referred to in the literature. Bruttin recorded an aneurysm of the internal carotid artery associated with unilateral papilledema. Loeb described a case of fracture of the skull in which this complication was present, and Kraft, a case of diffuse melanosarcoma involving all the meninges and penetrating into the smallest crevices of the skull and brain. For reasons to be discussed later, any space-taking lesion or any disease which raises the intracranial pressure may result in unilateral papilledema. One other cause, inflammation, might be mentioned here. Decking described a case of unilateral edema of the nerve head following the injection of alcohol into the gasserian ganglion. The patient also presented signs suggestive of a tumor of the pontile angle, so that the exact cause of the edema is not certain but may have been the result of a localized meningitis. Unilateral involvement is occasionally observed in meningitis.

Case 5 (service of Dr. Francis C. Grant).—J. D., a man aged 55, was admitted to the hospital on July 25, 1936. The chief complaint was headache behind the left eye of increasing severity for eight weeks. There was gradual extension to the back of the right eye. Personality changes occurred over a period of one year. The patient was stuporous on admission. Physical examination gave negative results. The cerebrospinal fluid pressure was 24 mm. of mercury. The

laboratory studies gave negative results. Roentgen examination showed erosion of the right frontal bone. Ophthalmoscopic examination of the right eye showed papilledema of 2 D. There was slight engorgement of the veins in the left eye but no papilledema. On July 25 a bone flap was turned down in the right frontoparietal region, and a subcortical tumor was found occupying the posterior part of the right temporal lobe and the anterior part of the right parietal lobe low down below the level of the sylvian fissure. A diagnosis of blastoma multiforme was made.

Case 6 (service of Dr. C. H. Frazier).—S. P., a man aged 47, was admitted to the hospital on Oct. 26, 1935. The chief complaints were headaches of increasing severity, of one month's duration, more marked in the right frontal region; increasing general weakness; apathy; impaired memory, and tinnitus on the right side. The blood pressure was 150 systolic and 80 diastolic. Physical examination revealed anosmia, a decrease in the tendon reflexes on the left side and weakness of the left side of the face. Ophthalmic examination showed a yellowish secretion in the conjunctival sac of the left eye. There was 4 D. of papilledema in the fundus of the right eye. The margins of the disk of the left eye were hazy, but there was no papilledema. Roentgen examination of the skull gave negative results. On October 31 a transfrontal craniotomy revealed a tumor in the right frontotemporal region, an astrocytoma.

Case 7 (service of Dr. C. H. Frazier).—W. C., a man aged 55, was admitted to the hospital on April 22, 1931. The chief complaints were vertigo; unsteadiness on standing or walking, slowly increasing since the onset in 1926; tinnitus in the left ear; falling to the right, and dull headaches. The blood pressure was 120 systolic and 80 diastolic. There was a positive Romberg sign. Ophthalmic examination showed lateral nystagmus when the patient turned the eyes to the extreme left. The visual fields were normal. Examination of the fundus of the right eye showed papilledema of 2.5 D. The fundus of the left eye was normal. Laboratory and roentgen studies gave negative results. A diagnosis of a tumor of the left cerebellopontile angle was made. Suboccipital craniotomy on May 9 revealed a neurofibroma of the eighth nerve.

Case 8 (services of Drs. T. B. Holloway and C. H. Frazier).—M. B., a girl aged 19, was admitted to the hospital on Sept. 25, 1935. The chief complaints were blurred vision in the right eye and vomiting on awakening for one month. On August 29 the patient had a fainting attack, followed by rigidity of the arms and partial loss of vision in the right eye. On September 22 she had an attack of unconsciousness, followed by a severe frontal headache. The blood pressure was 185 systolic and 100 diastolic. The patient suffered from somnolence. Physical examination revealed impairment of the muscle tone, hypoactive reflexes on the right side, numbness of the right fourth and fifth fingers and loss of sense of smell. Laboratory studies gave negative results. Roentgen examination showed increased intracranial pressure and erosion of the posterior clinoid processes. On ophthalmic examination the margins of disk of the right eye were found to be hazy; the color was yellowish but the disk was not atrophic. The right eye deviated outward 30 degrees, and there was amblyopia. Papilledema of 5 D. was present in the left eye. On October 7 a ventriculogram showed a tumor involving the third ventricle and obstructing the anterior portion. Right transfrontal craniotomy revealed a tumor of the third ventricle, an astrocytoma, blocking the outlet of the right lateral ventricle.

CASE 9 (reported by permission of Dr. Francis C. Grant).—E. G., a woman aged 40, complained of visual disturbance for four months, attacks of diplopia lasting from ten to fifteen minutes and occasional blurring of vision. Physical examination revealed impairment of the sense of smell on the right side, slight weakness of the lower right side of the face and deviation of the tongue to the right. Ophthalmic examination showed vision of 6/12 in the right eye and 6/9 in the left eye, with horizontal nystagmus on gaze to the left. The fundus of the right eye was normal. There was papilledema of 5 D. in the left eye. The visual fields were normal. A ventriculogram, made on Feb. 15, 1933, showed the ventricles displaced to the left. The right ventricle was deformed, and the anterior right horn was absent. Right transfrontal craniotomy revealed a tumor of the right sphenoid ridge, a fibroblastoma.

(*c*) *General Systemic Causes*.—Unilateral papilledema may theoretically occur in any disease which produces increased intracranial pressure. The mechanism will be discussed under the pathologic physiology. Adler reported a case of multiple sclerosis in which there was unilateral edema of the nerve head lasting for three months. It then disappeared completely and was followed by pallor in the temporal region. Shortly thereafter the same process was repeated in the other eye over the same period of time. The case is unusual, because of the unilaterality of the condition and also because papilledema is usually evanescent in multiple sclerosis. Brazeau described a case of chlorosis with unilateral involvement. Syphilis is probably the most frequent cause in this group, although there are no statistics to support such a belief. The papilledema due to systemic disease owes its unilaterality in most instances to localized changes in the eye, orbit or cranium.

CASE 10 (services of Drs. Francis C. Grant, John H. Stokes and Francis Heed Adler).—M. D., a man aged 27, was admitted to the hospital on May 6, 1937. The chief complaint was marked diminution of vision in the left eye of three weeks' duration. The patient had an initial syphilitic lesion in January 1937. He was treated at another hospital and received four weekly injections of an arsenical preparation. No therapy was given during February and March. During the first week in April the patient received one intravenous injection of some drug, followed by diminished vision in the left eye. He then went to a second hospital, where he was said to have had papilledema of 4 D. in the left eye and slight papilledema in the right eye. While there, he was suspected of having a tumor of the brain and was sent to the Hospital of the University of Pennsylvania. Physical examination gave negative results. The Wassermann reaction of the blood was strongly positive. The spinal fluid gave a negative reaction to the Wassermann test; the protein content was 75 mg. per hundred cubic centimeters, the cell count 10 and the colloidal gold curve 0122221000. External examination of the eye gave negative results. Examination of the fundus of the right eye showed the outlines of the disk to be blurred and the disk to be slightly elevated, possibly 1 D. There was papilledema of 4.5 D. in the fundus of the left eye, with many hemorrhages and a macular star figure. Vision was 6/9—2 in the right eye and 1/60 in the left eye. The visual field of the right eye showed slight concentric contraction and an enlarged blindspot; that of the left eye showed slight concentric contraction and marked enlargement of the blindspot. The patient

was put on a course of antisyphilitic therapy. On October 19 examination of the fundus of the right eye showed the disk to be still slightly elevated, possibly 0.5 D. The fundus of the left eye showed papilledema of 3 D., slight edema of the macula and a slight pigmentary disturbance. Vision was 6/12—2 in the right eye and 6/150 in the left eye. The visual field of the right eye showed concentric contraction; the field of the left eye was similar.

This case is included because the papilledema of the right eye was negligible after antisyphilitic therapy. There is some doubt as to whether the process in the left eye was pure papilledema because of the low vision. This, however, may be accounted for by the fact that the edema extended out to the macula. The visual fields were more suggestive of papilledema than papillitis, although the latter cannot be definitely excluded. The fact that only one nerve head showed much edema could be accounted for by a localized arachnoiditis around the right optic foramen, which prevented the cerebrospinal fluid from entering the intervaginal space of the right optic nerve.

Case 11 (service of Dr. Francis C. Grant).—V. C., a man aged 29, was admitted to the hospital on Feb. 1, 1937. His chief complaints were headaches of one month's duration, occasional nausea and vomiting of increasing frequency and severity and, later, failing memory for recent events, loquacity, vertigo and blurred vision in the left eye. Physical examination showed slight weakness of the mouth on the right side and hyperactive patellar reflexes, more marked on the right. On ophthalmic examination it was found that the pupils did not react to light or in accommodation and that the corneal reflexes were hyperactive. Examination of the fundus of the right eye showed possible slight edema of the nerve head but no measurable elevation. Papilledema of 3.5 D. was present in the left eye. The visual fields showed a right homonymous superior sector cut and either a binasal inferior sector cut or possibly a left homonymous inferior defect with a reentering angle about 15 degrees left of the midline, breaking through to the blindspot in the left eye and also catching the lower nasal field in that eye. The blindspots were greatly enlarged. Vision was 6/9 in the right eye and 3/60 in the left eye. The cerebrospinal fluid pressure was 27 mm. of mercury. The Wassermann reaction of the spinal fluid was negative, and the colloidal gold curve was 1122244444. Roentgen examination revealed a possible tumor of the right sphenoid ridge. On February 27 a ventriculogram showed internal hydrocephalus with evidence of a block in the proximal portion of the iter. On suboccipital craniotomy tonsils of cerebellum were found adherent to the opening of the fourth ventricle. The tonsils and vermis were freed of adhesions. Sections of tissue were taken from about the opening of the fourth ventricle. Microscopic examination revealed syphilitic meningitis. Necropsy disclosed syphilitic basilar arachnoiditis.

The mechanism in the case, which limited the papilledema to one eye, was probably the same as that suggested in the preceding case.

Case 12 (reported by permission of Dr. Francis Heed Adler).—B. M., a man aged 45, was first seen in December 1926. Vision in each eye was 6/6. The fundi were normal. On June 27, 1931, the patient had an attack of glaucoma in the right eye. The tension (Schiötz) in the right eye was 60 mm. of mercury

and in the left eye, 13 mm. The tension was controlled with miotics until March 12, 1935, when the Elliot trephine operation was performed on the right eye. On March 27 the tension (Schiötz) in the right eye was 9 mm. of mercury and in the left eye 18 mm. About this time signs and symptoms of an arterial hypertension began to develop. The intraocular tension in the right eye began to drop steadily, and on June 14 it was 5 mm. of mercury in the right eye and 17 mm. in the left eye (Schiötz). No ocular signs of hypertension developed until October 1, although the patient was seen at least once a month. At that time the fundus of the right eye showed arteries deeply indenting the veins, which were markedly overfilled, and several small points, which were suggestive of hemorrhages. The fundus of the left eye was normal. The tension (Schiötz) in the right eye was very low; that in the left eye was 18 mm. of mercury. The blood pressure was 180 systolic and 100 diastolic. On October 7 there was no change except for a definite hemorrhage on the right disk. The left eye was normal. On December 5 the tension in the right eye was extremely low, and the eye was soft. The tension (Schiötz) in the left eye was 21 mm. of mercury. Examination of the fundus of the right eye showed many hemorrhages on the disk, marked arteriovenous compression and a definite edematous appearance. The fundus of the left eye showed slight arteriosclerotic changes, normal for the patient's age; otherwise the fundus was normal. On Jan. 1, 1936, the tension in the right eye was extremely low on palpation; that in the left eye (Schiötz) was 25 mm. of mercury. The fundus of the right eye showed papilledema of from 2.5 to 3 D., many hemorrhages and cotton wool exudates and marked arteriovenous compression. The fundus of the left eye was normal. On October 29 the tension (Schiötz) in the right eye was 7 mm. of mercury and in the left eye 21 mm. There was little change in the fundi. The blood pressure was 190 systolic and 125 diastolic. On Jan. 7, 1937, the tension (Schiötz) in the right eye was 11 mm. of mercury and in the left eye 21 mm. There were no hemorrhages or exudates in the fundi and little edema of the nerve heads. On March 12 the tension (Schiötz) was 12 mm. of mercury in the right eye and 25 mm. in the left eye. Examination of the fundus of the right eye showed tortuous vessels and an increase in light streaking. On January 5 the blood pressure was 210 systolic and 130 diastolic. On palpation the tension was normal in each eye. The fundi showed no change. On March 25 the blood pressure was 190 systolic and 120 diastolic. The tension (Schiötz) was 14 mm. of mercury in the right eye and 23 mm. in the left eye. The fundus of the right eye showed well marked arteriosclerosis. The disk had a good color, and there was no papilledema, hemorrhages or exudates. The fundus picture of the left eye was the same.

The exciting cause for the papilledema in this case was probably the lowered intraocular pressure. It is interesting to note that although the patient had all the signs and symptoms of hypertension, marked changes did not develop in his left eye. The arterial hypertension steadily increased, but as soon as the intraocular tension of the right eye increased, the papilledema, cotton wool exudates, hemorrhages and marked venous dilatation disappeared. One school of thought believes that retinal changes of this type seen in patients with arterial hypertension are definitely indicative of a serious prognosis and that only a small percentage of such persons survive for more than a few years. There are many persons who have shown such changes and are still in fair health

many years later. It is within reason to say that in this case the retinal changes which accompanied arterial hypertension were markedly accentuated by the low intraocular tension. It is well known that such patients may show remissions. The disappearance of the retinal changes of the right eye was probably not an ordinary remission for two reasons. In the first place, the arterial tension rose constantly. In the second place, the disappearance occurred only after an increase in the intraocular tension of this eye. Moreover, no similar change took place in the left eye. There is, therefore, a direct relationship between the degree of retinal change and the ocular tension. It is probable that the evaluation of any case in which there is marked arterial hypertensive changes may be considerably more accurate if the intraocular tension is taken into account. As far as I know, this point has hitherto not been mentioned.

PATHOLOGIC PHYSIOLOGY

In order to understand the mechanism of unilateral papilledema, one must have a clear conception of the physiologic changes which may take place. The most widely accepted theory of the cause of papilledema is probably that of Paton and Holmes. According to these authors, the primary cause of the edema is pressure on the vena centralis in the intervaginal space. Fry studied an extensive series of cases of papilledema by means of serial sections and found ample evidence to show that the compression of the central vein of the retina takes place either along the side of the nerve or in the subarachnoid space. In another study he found that there is considerable variation in the course of the central artery and vein. He stated: "The relatively long course of the vein in the subpial and intervaginal space . . . would favor compression of this vessel by increased pressure of the fluid within the dura in cases of increased cerebrospinal fluid pressure, and hence be of importance in the production of papilledema." He was able to show that "the secondary cause of papilledema is a forward pressure of fluid within the optic nerve produced in all probability by the infiltration of spinal fluid under increased pressure in the perivascular lymph spaces." One may then say that papilledema is at least in part due to changes in the normal circulation through the central retinal vein and that unilateral papilledema may be the result of factors influencing the circulation through one central retinal vein. The simplest interference resulting in unilateral papilledema is pressure directly on the vein, such as may arise from intraorbital causes, tumors, cysts, aneurysms and periostitis. In such cases there are often other concomitant symptoms and signs, such as exophthalmos and involvement of the orbital nerves, which aid in diagnosis.

The mechanism in unilateral papilledema due to an increase in intracranial pressure is somewhat more complicated. First of all one disk may be free from papilledema because no fluid, or only a small amount, gains access to the intervaginal space from the cranial vault. This may be due to arachnoiditis or some other localized process, such as tumor growth in the region of the optic foramen, which blocks the exit of fluid from the skull. Moreover, as Marchesani and others have pointed out, papilledema usually occurs only in vital nerves. In 1 of his cases there was edema only on the nasal side of the disk and none in the atrophic temporal portion. The same author described a second case in which there was papilledema of several diopters in one eye as a result of increased cerebrospinal fluid pressure. The second eye showed no papilledema. Ophthalmoscopic examination showed 20 D. of myopia and an atrophic nerve head. A careful necropsy and histologic studies showed typical papilledema on the one side and only a faint suggestion thereof in the atrophic nerve. These two factors, atrophy and blockage at or near the optic foramen, are of importance in the syndrome described by Gower, Paton, Uthoff, Traquair, Foster Kennedy and others. In this syndrome of tumor of the frontal lobe there is atrophy of the optic nerve on the side of the tumor and papilledema on the other side. It must not be assumed, however, that given a case of tumor of the frontal lobe with atrophy on one side and papilledema on the other that one is necessarily confronted with a tumor located on the side of the atrophic disk. Dowman and Smith reported a case in which there were papilledema on the right side and atrophy on the left side. The tumor was located in the right frontal lobe. Thies and others have described similar cases. It may be that in these cases there was atrophy of one nerve of long standing due to an entirely different cause. Another explanation must be considered. The increased intracranial pressure may have caused unilateral papilledema at first, which progressed to atrophy. After this, the intracranial pressure steadily rose and caused papilledema in the other nerve. This mechanism will be considered more in detail later.

In the absence of the two aforementioned factors, unilateral papilledema may still occur in intracranial hypertension. As Fry has shown, the central retinal vein may run through the intervaginal space for some distance. If one central vein were exposed to the pressure of cerebrospinal fluid for a greater distance than the other, unilateral papilledema might occur. The longer the course of the vein in the intervaginal space, the less will be the pressure needed to produce papilledema in the corresponding nerve head.

Increased cerebrospinal fluid pressure may result in unilateral papilledema in still another manner. All that is needed is a rise in the

pressure in one central retinal vein. The return venous blood from the ophthalmic veins passes through the two cavernous sinuses, which are joined anteriorly and posteriorly by the annular sinuses. Leaving these, a large volume of blood passes to the junction of the transverse and sigmoid sinuses by way of the superior petrosal sinuses. Some of the blood also passes from the cavernous sinuses downward and posteriorly by way of the inferior petrosal sinuses to the jugular bulb. The sigmoid sinuses run from the junction of the superior petrosal and transverse sinuses to the jugular bulb. The transverse sinuses are fed by the large central sinuses, which drain both cerebral hemispheres, the midbrain and the cerebellum, through the superior and inferior longitudinal and straight sinuses. Swift, in a study of the relation of the cranial sinuses to papilledema, showed how tumors may change the venous pressures. It may be that pressure on the right cavernous and inferior and superior petrosal sinuses could cause a rise in the pressure in the right ophthalmic vein. Pressure on the right transverse sinus could cause a rise in the pressure in the left ophthalmic vein. Thus unilateral papilledema could be produced. Gibbs pointed out how such a disturbance might cause unequal edema in the optic nerve heads in tumors of the brain. In most cases unilateral papilledema due to increased intracranial pressure progresses to bilateral involvement, and it is not unreasonable to presume that in most cases of unequal bilateral papilledema the condition began with involvement of only one disk. It is highly problematic whether interference with circulation of the sinuses alone, in the absence of a generalized increase in intracranial pressure, could cause papilledema, as evidenced by the number of cases of sinus thrombosis in which there was no papilledema (Uhthoff). As Gibbs has pointed out, this theory needs much carefully controlled experimental work. Many factors, such as the rapidity and direction of tumor growth relative to the sinuses, are not taken into consideration here. Moreover, as Swift and others have pointed out, the cranial sinuses vary enormously in size, location and anastomoses. All these factors must influence the development of papilledema. The circulation of the sinuses is probably quickly adjusted to any interference at one point or another, so that this factor by itself is not in all likelihood of cardinal importance.

The physiologic mechanism of unilateral papilledema of ocular origin is more abstruse. According to Sobański and Lauber, an increase in the diastolic venous pressure and its relation to the arterial diastolic pressure in the retina are decisive factors in the production of papilledema. The work of these authors shows that any alteration in the ratio of these pressures, as measured by their methods, beyond 1 : 1.5 always leads to papilledema. It is likely that this relation is somehow or other disturbed in cases of papilledema due to low intraocular pressure. Paton and Holmes, Kyrieleis, Parker and others have shown that the eye with the

lower intraocular pressure shows the greater degree of papilledema in cases of increased cerebrospinal fluid pressure. The lamina cribrosa may be considered as a diaphragm which moves forward with an increase in the intracranial pressure (Fry). In the event that the cranial pressure remains the same, the lamina cribrosa might move forward if the resistance against the normal intracranial pressure were removed, as is the case when the intraocular tension is lowered. The normal lamina cribrosa is convex toward the optic nerve and concave toward the lens. If it is pushed anteriorly, the structures passing through its interstices must be compressed. The vein being more easily collapsible than the artery, because of its thinner walls, would suffer more compression. Such compression would raise the venous pressure, so that edema might result or at least be increased. In an eye in which the pressure had been reduced by means of a cyclodialysis for glaucoma, Sobański and Lauber found papilledema of 2.5 D. The diastolic arterial pressure was 38 mm. of mercury and the diastolic venous pressure 25 mm., a ratio of 1.5 : 1.

SUMMARY

The causes of unilateral papilledema may be grouped under the following headings: intraocular, intraorbital, intracranial and systemic. The last is probably dependent on localized changes in the region of the optic foramen or to a combination with one of the other causes.

In the event of a space-taking lesion the papilledema may not be homolateral and cannot be depended on by itself to determine the side on which the tumor is located.

Patients presenting unilateral papilledema should be subjected to a study, including measurement of the intraocular pressure, exophthalmometer measurement, thorough neuro-ophthalmic examination, spinal fluid studies and roentgen examination of the head, including the optic foramens and the sinuses.

The importance of certain factors which have not received due consideration heretofore is discussed. These are: the relation of intraocular pressure to papilledema, particularly in arterial hypertension, and the yielding of the lamina cribrosa, with direct compression of the vein and elevation of the retinal venous pressure.

339 North Duke Street.

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News and Notes

SOCIETY NEWS

Association for Research in Ophthalmology.—The tenth scientific meeting of the Association for Research in Ophthalmology, Inc., will be held on May 16, 1939, at the Coronado Hotel, St. Louis. The following papers will be presented: "Experimental Ocular Hypersensitivity," by Dr. T. E. Sanders, department of ophthalmology, Washington University, St. Louis; "Studies on Surface Epithelium Invasion of the Anterior Segment of the Eye," by Drs. T. L. Terry, J. F. Chisholm and A. L. Schonberg, Massachusetts Eye and Ear Infirmary, Boston; "A Study of Methemoglobin-Producing Organisms in Ocular Inflammations," by Dr. Maynard A. Wood, department of ophthalmology, University of Iowa, Iowa City; "The Relation of Müllers Orbital Muscle to the Pathology of Retrobulbar Tissues Obtained in Experimentally Produced Exophthalmos," by George K. Smelser, Ph.D., department of ophthalmology, Columbia University, New York; "Ocular Reactions of Horses and Rabbits Infected with Strains of Brucella Recovered from Horses with Periodic Ophthalmia," by Dr. E. L. Burky, Robert Redvers Thompson, Ph.D., and Helen D. Zepp, A.B., the Johns Hopkins Hospital, Baltimore; "Staphylococcus Conjunctivitis: Experimental Reproduction with Staphylococci," by Dr. James H. Allen, department of ophthalmology, University of Iowa, Iowa City; "An Immunological Study of Trachoma," by Louis Julianelle, Ph.D., department of ophthalmology, Washington University, St. Louis, and "Vitamin-D Complex in Myopia: Etiology, Pathology and Treatment," by Dr. Arthur E. Knapp, New York.

Oxford Ophthalmological Congress.—The Oxford Ophthalmological Congress will assemble at Keble College, Oxford, on Wednesday evening, July 5, and the meetings will be held on the following Thursday, Friday and Saturday, July 6, 7 and 8.

The Doyne Memorial Lecture will be delivered by Mr. F. A. Williamson-Noble, F.R.C.S., of London, England, and will be entitled "Ocular Consequences of Certain Chiasmal Lesions."

On Thursday morning, July 6, there will be a discussion on orthoptic training. The discussion will be opened by Mr. Frank Law and Mr. Keith Lyle, who will be followed by Miss Sheila Mayou, Messrs. E. B. Alabaster, George Black, H. Campbell Orr, P. Jameson Evans, Bernard Chavasse and O. M. Duthie.

On Saturday morning, July 8, Mr. Harrison Butler will open a discussion on miners' nystagmus.

The following papers have already been promised: "The Latest Principles for Applying Tinted Glass to Industrial and Other Purposes," by Sir Arnold Lawson, of London; "Drusen of the Retina: A Clinical and Pathological Study," by Dr. Bernard Samuels, of New York; "Number Forms," by Mr. P. J. Doyne, of London, and "The Ophthalmic Surgeon's Functional Operative Paresis," by Mr. Basil Graves, of London.

GENERAL NEWS

American Board of Ophthalmology.—The American Board of Ophthalmology will conduct a written examination in various cities in the United States, in Honolulu, Territory of Hawaii, and in Puerto Rico, as well as in Canada, on August 5. Formal application for this examination must be received before July 1. General oral examination for successful candidates will be held on October 7 in Chicago.

For application blanks and information, write at once to the secretary, Dr. John Green, 6830 Waterman Avenue, St. Louis.

Abstracts from Current Literature

Edited by Dr. William Zentmayer

Biochemistry

The Phosphatases of the Lens. A. Santoni, Ann. di ottal. e clin. ocul. **66:** 209 (March) 1938.

An extract of whole beef lens was allowed to react with a standard solution of sodium glycerophosphate. The material was examined for the amount of phosphorus present after deproteinization with trichloroacetic acid, and this amount was compared with the phosphorus in the lens extract alone. Splitting of the phosphate occurred best at p_H 9.2. From 4 to 12 per cent of the phosphate present in the solution was broken down in twelve hours, indicating definitely the presence of phosphatase in the lens. The amount is less than that found in most other tissues.

S. R. Gifford.

Blindness

Causes of Blindness, editorial, Brit. M. J. **2:** 796 (Oct. 15) 1938.

The thirty-second annual report of the Northern Counties Association for the Blind presents an analysis of 10,000 cases of blindness, made by the medical subcommittee, of which Dr. Kay Sharp, consulting ophthalmic surgeon, West Riding County Council, is chairman.

Of the 10,000 cases of blindness, 6,406 were due to congenital or undetermined causes, primary cataract accounting for more than one-half and primary glaucoma for one-sixth. The number of cases due to infectious and bacterial causes was 1,791, of which 405 were ascribed to ophthalmia neonatorum and 492 to syphilis, congenital or acquired.

There were 580 cases of blindness due to traumatic and chemical causes, industrial trauma accounting for 262 and nonindustrial trauma for 286.

Eight hundred cases of blindness were ascribed to general disease, the greatest number being due to vascular diseases, including cerebral vascular lesions, and the next greatest number, to diabetes.

Of the 10,000 cases, no information as to cause was obtainable concerning 423.

Arnold Knapp.

Color Sense

Behavior of the Color Sense During Compression of the Globe. A. Santonastaso, Ann. di. ottal. e clin. ocul. **66:** 161 (March) 1938.

When pressure is made on the globe during observation of a colored target, two phenomena occur. First, the color of the object is lost, and as pressure is increased, the object itself is lost to view. The author investigated these phenomena in 10 normal observers with blue, red and green targets observed in natural light and with lights of pure spectral color in the darkroom after suitable adaptation. Observations

for these colors were made not only at the fixation point but at various points on the horizontal meridian, including 40, 25 and 10 degrees on the temporal side and 25 and 15 degrees on the nasal side. The pressure of the dynamometer required to abolish the color and that required to abolish perception of the target were recorded, the difference in the two pressures measuring what the author calls the chromoluminous interval. The results were fairly constant for all the persons examined. The results, in general, were as follows:

1. For the central portion of the retina, less pressure was necessary to obliterate the color in the darkroom than in daylight, while for the peripheral portion of the retina the opposite was the case. This indicates that the peripheral portion of the retina functions better than the central portion in the darkroom, while in daylight the central portion is superior.

2. The chromoluminous interval was greater, especially for red and blue, in the periphery than in the center.

3. When the intensity of the colored stimulus was decreased, less pressure was necessary to obliterate it.

4. In the extreme periphery less pressure was necessary to obliterate the color. This was especially true at the nasal side and with observation in daylight.

5. When the color disappeared during pressure, a slight movement of the target caused it to reappear, as a rule, but this was more uniform if it was moved toward the fixation point. If it was already at the fixation point, a slight movement to either side caused it to reappear, but when high pressures were employed, this sometimes occurred only when it was moved to the temporal side.

For other interesting details and some conclusions bearing on the theories of color vision, the original article must be consulted. Perhaps the most important of these is one suggested by the fact that dark adaptation did not improve the function of the retina for color so far as to increase the pressure required to obliterate color sensation even in the retinal periphery. A bibliography accompanies the article.

S. R. Gifford.

Conjunctiva

Primary Tuberculous Infection of the Conjunctiva. Benalioua, Ann. d'ocul. **175**: 746 (Oct.) 1938.

Primary tuberculous infection of the conjunctiva, the existence of which has been demonstrated, is a disease of children and young adults and is seen more frequently in females than in males. It is manifest especially as a unilateral conjunctival pretragal complex, of a sluggish nature, associated with various lesions of the mucous membrane and frequently with numerous caseous glands.

The prognosis of this condition, long considered particularly grave, is in reality favorable, cures occurring without any complication except bacillary migrations, which are without danger and are no doubt conveyed by an erythema. The relatively good prognosis permits primary

tuberculous infection of the conjunctiva to be placed under the same pathogenic classification as visceral tuberculosis and particularly pulmonary tuberculosis.

Treatment of the ocular condition should not be too aggressive, and it is considered much the best plan, as in a primary pulmonary infection, to institute general tonic systemic treatment and to reserve the usual local treatment for the form of slow evolution with a manifest tendency to spontaneous relapse.

A bibliography of four pages accompanies the article.

S. H. McKee.

Fate of Patients with Phlyctenular Disease with Particular Refference to Tuberculosis. A. Ajo, Acta ophth., 1938, supp. 15, p. 1.

This monograph first gives a historical review of ideas which have been held in regard to the etiology and pathogenesis of phlyctenular disease. It then deals with the question of the prognosis of ocular phlyctenulosis with particular regard to the later development of tuberculosis.

In the University Eye Clinic at Helsingfors, Finland, there were 449 patients with phlyctenular disease between 1912 and 1927. The author succeeded in tracing 367 of these patients. He found that 55 had died. Tuberculosis was the cause of death of 35, and for 4 more it was the probable cause, making the mortality rate for tuberculosis 10.62 per cent. This is about double the mortality rate for tuberculosis for persons of the same age group in the general population of Finland.

Of the former patients still living, 100 were thoroughly examined. Fifty were found free from disease. Thirty-nine of the remaining 50 patients showed tuberculous changes. Five showed changes which were probably tuberculous and 6, other pathologic changes. Pulmonary tuberculosis was found in 7 patients. The author concludes that it may not be held that patients with phlyctenular disease will be spared later severe tuberculosis.

O. P. Perkins.

Congenital Anomalies

Aplasia of the Optic Nerves. H. Ridley, Brit. J. Ophth. 22:669 (Nov.) 1938.

This rare anomaly was seen in a child 1 year of age. The child did not follow light. There was no nystagmus. The anterior portions of the eye were normal. The pupils were inactive to light but varied in size from time to time, irrespective of stimulus. The media were clear. The fundus reflex was pale red. In place of the optic nerve head there was a deep excavation, the floor of which was about 5 diopters beyond the level of the fundus. Its color was gray. From the floor of the pit emerged only minute threads, representing retinal vessels, the lower branch in the right eye being the only one which contained more than a trace of blood, and even this appeared to fade out soon after leaving the edge of the excavation.

The article is illustrated.

W. Zentmayer.

General Diseases

BERIBERIC RETROBULBAR NEURITIS. F. J. SORIANO and M. I. PUIGGARI, Arch. de oftal. de Buenos Aires **13:** 244 (May) 1938.

After reference to the ocular symptoms connected with avitaminosis A, C and D, the authors consider avitaminosis B. They discuss at considerable length the production of this type of avitaminosis, observed mostly in persons eating shelled rice.

Avitaminosis B attacks the motor, sensory and optic nerves, and appears in two forms: (1) a dry, or paralytic, form, with marked muscular atrophy and some anesthesia, and (2) an edematous, or dropsical, form, with generalized edema and violent pain.

The manifestations due to involvement of the ocular nerves are numerous and frequent, and all the ocular nerves may be involved. The optic nerve is frequently affected; according to Japanese writers, it is usually affected. Involvement of this nerve appears as retrobulbar neuritis with a central scotoma and atrophy of the papillomacular bundle. The scotoma is characterized by a scotomatous bridge joining the scotoma proper with the blindspot.

The author reports a case in which retrobulbar neuritis was the sole symptom of beriberi.

A woman who had suffered great privations in Spain, having subsisted for some time on an almost exclusive diet of rice, with no meat, fat or milk, suffered from loss of sight and loss of 20 kilograms of weight. There was a central scotoma of the type described. The use of alcohol and tobacco could be excluded as well as the use of hair dyes. There was no diabetes, and the blood and urine were normal.

The authors consider the prognosis poor as regards a permanent improvement of vision, as they believe the lesion to be stationary.

C. E. FINLAY.

Glaucoma

CYCLODIALYSCLERECTOMY: ANTIGLAUCOMATOUS OPERATIONS. E. GORNET, Ann. d'ocul. **175:** 678 (Sept.) 1938.

As the title implies, this operation consists in uniting sclerectomy and cyclodialysis into one operation. It is not a question of denying the good effect of a limbic sclerectomy or of cyclodialysis but a question of attempting to correct the failures of one or the other operation in the association of the two.

The author describes the two operations minutely.

A bibliography accompanies the article.

S. H. MCKEE.

TREATMENT OF ABSOLUTE PAINFUL GLAUCOMA BY ROENTGEN THERAPY. P. SATANOWSKY, Arch. de oftal. de Buenos Aires **13:** 227 (May) 1938.

After reference to, and a comparison of, various methods of treatment recommended for the relief of the intolerable pain in cases of

absolute glaucoma, such as opticociliary neurotomy, enucleation and retrobulbar injections of alcohol, the author reports 3 cases in which relief was obtained by roentgen therapy, a procedure first employed by Hessberg in 1920.

The literature on the subject is reviewed. C. E. FINLAY.

Injuries

KERATOCONJUNCTIVITIS FROM CHESTNUT BURS. C. DEJEAN and R. GUIGNOT, Arch. Soc. d. sc. méd. et biol. de Montpellier **19**: 70, 1938.

A dried chestnut shell on hitting a child's eye produced an irritation for over two months, with photophobia, lacrimation, diminished visual acuity, edema of the lids, chemosis and mucopurulent secretion. The slit lamp revealed from seven to eight punctiform leukomas of rusty color, probably representing the spicules of the chestnut bur. Fortunately the débris was eliminated spontaneously, with considerable visual recovery.

J. E. LEBENSOHN.

TWO STAGE ROENTGEN OBSERVATION DURING OPERATION FOR INTRAOCULAR FOREIGN BODIES: REPORT OF CASES. R. TERTSCH, Klin. Monatsbl. f. Augenh. **100**: 339 (March) 1938.

Tertsch describes a new method for removing foreign bodies of weak magnetic or nonmagnetic power from the eyeball. His method is based on the principles reported by George H. Cross in 1927. Tertsch refers to the excellent work done in this realm by von Szily during the World War. The method, described in detail, centers in the use of two roentgen spheres in connection with a superastral screen. Two tubes are fitted to the spheres so as to limit irradiation to a circular opening with a diameter of 30 mm. The tubes contain a filter which allows illumination for several minutes without injuring the skin. The two superastral screens, with a surface of about 5 sq. cm., are fastened to a handle. An appliance is being prepared which will permit fastening the screen to the patient's head. The two bundles of roentgen rays unite in the region of the foreign body at an angle of approximately 90 degrees in a manner which will furnish two pictures situated perpendicularly to one another. Interchanging observation of the two pictures allows orientation and depth vision, enabling the operator to approach the foreign body readily with his instrument. Lesion of the healthy eye is rendered impossible by proper devices. The roentgen spheres are focused by pedals worked with the foot. The room is darkened, and the operator wears a head light sufficient to illuminate the operative field. Its weak light does not interfere with the luminescence of the roentgen pictures. A detailed description of the instruments is given, and the use of the method is illustrated by 3 cases in which operation was successful.

K. L. STOLL.

Lacrimal Apparatus

LACRIMAL LAMINARIA IN THE SURGICAL THERAPY OF ATRESIA OF THE NASAL CANAL. P. ZARZYCKI, Bull. Soc. d'opht. de Paris **50:** 306 (June) 1938.

In gynecology Laminaria (seatangle) renders a great service. In ophthalmology, because of blunders of technic, it has been neglected. It is superior to metal sounds, which cause trauma to live tissue and necessitate frequent treatments. It is more rigid than the fiber sound and has the advantage of increasing its bulk slowly over a period of hours. Its use is contraindicated in cases of osteitis, tuberculosis or syphilis affecting the region of the lacrimal sac, obstruction due to foreign body, exostoses and polyps. Zarzycki outlines the technic for the use of seatangle. The details given are too numerous and lengthy to be recorded here.

L. L. MAYER.

Lens

HEREDITARY DEGENERATION OF THE RAT RETINA (ASSOCIATED WITH CATARACT). M. C. BOURNE, D. ADAMS CAMPBELL and KATHARINE TANSLEY, Brit. J. Ophth. 22: 613 (Oct.) 1938.

This paper deals with a study of the retina of rats with hereditary cataractous lenses. Briefly, the changes in the retina were of the nature of progressive degeneration beginning with the death of the rod nuclei. The subsequent alteration in the structure of the retina leading ultimately to disintegration of the inner nuclear and ganglion cell layers appears to be the result of the disappearance of the neuroepithelium. There were no gross vascular changes in the retina, and the blood vessels of the choroid remained unaffected until an advanced stage of retinal degeneration. The authors were impressed by the striking resemblance which the histologic picture of this lesion in certain stages of development bears to the microscopic appearance described in certain cases of retinitis pigmentosa.

The article contains a number of illustrations.

W. ZENTMAYER.

Lids

THE GENESIS OF EPITARSUS. G. ANASTASI, Ann. di ottal. e clin. ocul. **66:** 202 (March) 1938.

A man, aged 22, showed symmetric folds of conjunctiva near the upper border of both upper tarsi. A probe could be passed upward beneath each fold into the retrotarsal folds. The conjunctiva and eyes were otherwise normal. Purulent conjunctivitis had been present in both eyes at birth, and the author believes that the condition was caused by applications of the silver nitrate stick. It was, therefore, simply a peculiar form of symblepharon and not a congenital anomaly.

A bibliography accompanies the article.

S. R. GIFFORD.

Neurology

OCULAR ORIGIN OF MIGRAINE IN CHILDREN AND YOUNG PERSONS. K. GRUNERT, München. med. Wchnschr. **85:** 1337 (Sept. 2) 1938.

For ten years Grunert made careful functional tests on the eyes of all patients who were subject to headaches and to migraine and, as far as possible, of the relatives of these patients. In the course of these studies he made two significant observations:

1. In the majority of the patients with headaches and in nearly all of those with migraine there existed disturbances in the ocular function.

2. The treatment of these ocular disturbances reduced or completely removed the general symptoms to such an extent that a causal connection could not be denied. In children and young persons the ocular origin of migraine was even more pronounced than it was in adults.

The author reports observations on more than 200 children and young persons with migraine. Many of the children were of the type referred to as "difficult"; that is, they showed signs of nervous irritability, were difficult to manage or were inattentive in school. Ocular examination disclosed defective refraction, disturbances in the ocular equilibrium and defects in accommodation. In many of these children the growth of the eyes is not completed as early as in others, and school, which requires a considerable amount of close work, involves a great strain on these still growing eyes. After describing some of the difficulties of these children and of the symptoms likely to develop in them, the author directs his attention to the treatment of migraine of ocular origin. He emphasizes that even slight optic defects may cause severe subjective disturbances. After the refractive defects have been corrected, close work is avoided for one or two weeks, and the weakened muscle of accommodation is treated with pilocarpine. The administration of pilocarpine must be individualized. It is best to administer it in the evening before the patient goes to sleep. In cases of severe defects it can be given also during the day, but the individual doses are, of course, smaller. In cases of disturbances in the muscular equilibrium or in latent strabismus prisms are employed; occasionally an operation for strabismus may be resorted to, or stereoscopic exercises may be employed. In the few cases in which no anomalies of refraction exist and in which there is no impairment of the muscular equilibrium but only an insufficiency of the apparatus of accommodation, the migraine is cured by resting the eyes for a while and by treatment with pilocarpine. The ocular treatment effected cure in 197 children and young persons with migraine. The author closes his report by recommending that all children in whom migraine is suspected be subjected to a thorough examination of the eyes.

J. A. M. A.

Ocular Muscles

RELATIONSHIP OF HETEROPHORIA TO DIVERGENCE AND CONVERGENCE, BASED ON CLINICAL MEASUREMENTS. F. H. HAESSLER, Am. J. Ophth. **21:** 272 (March) 1938.

Haessler discusses heterophoria and after extensive tests gives the following summary and conclusion:

"In 1,000 patients who came for refraction, it was shown that the degree of exophoria or esophoria was not determined by a balance between divergence and convergence as expressed in the usual clinical measurements."

W. S. REESE.

OPERATION IN A CASE OF MARCUS GUNN PHENOMENON. A. and G. OFFRET, Bull. Soc. d'opht. de Paris **50:** 61 (Feb.) 1938.

The family of a girl of 9 years insisted that an operation be done for ptsois of the right eyelid. The eyelid covered a greater part of the cornea and was accompanied by a limitation of elevation of the globe. In mastication, when the mouth opened the lid raised, only to return to the original position when the mouth was closed. The operation used has been described by Nida (*Ann. d'ocul.* **166:** *639, 1929*). It consists of an insertion of the tendon of the superior rectus muscle into the tarsus of the upper lid. Two and one-half years after operation the child's lid still remained motionless when the mouth was opened. There was also no movement of the lid on mastication. A bibliography accompanies the article.

L. L. MAYER.

Orbit, Eyeball and Accessory Sinuses

LUXATION OF THE EYEBALL INTO THE ANTRUM; ILLUSIVE ANOPHTHALMOS DURING AUTOPSY: REPORT OF A CASE. A. JESS, Klin. Monatsbl. f. Augenh. **100:** 353 (March) 1938.

After a brief review of the literature in point, the author reports the case of a woman, aged 64, who injured her eye by falling against the edge of a door. The conjunctiva was retracted funnel shape; blood oozed from the orbit, and a cloud was observed in the right antrum on roentgen examination. Operation revealed remnants of vitreous, sclera and finally the collapsed eyeball, which was removed with a stump of the optic nerve measuring 1 cm. After removal of a large blood clot from the antrum, the wound healed promptly. The eyeball had slipped into the antrum through a fracture of the floor of the orbit. In another case, reported to Jess, a soldier died from a fractured skull after falling off a roof. No eyeball was observed in one orbit during autopsy, so that the question arose as to whether the man wore a prosthesis and escaped detection during his military services. Although his wife persisted in stating that he had two good eyes, proof was lacking, as the body had been buried. Altercation with insurance companies are discussed in this respect. The cases referred to include 1 reported by Becker, that of a farmer girl who suffered subluxation of the eyeball into the antrum by the thrust of a cow's horn, and the case of the boy who could see through one nostril after a similar injury, which occurred in 1575. Both accidents happened in Heidelberg.

K. L. STOLL.

Pharmacology

HUMAN AUTONOMIC PHARMACOLOGY: XI. EFFECT OF BENZEDRINE SULFATE ON THE ARGYLL ROBERTSON PUPIL. ABRAHAM MYERSON and WILLIAM THAU, Arch. Neurol. & Psychiat. 39:780 (April) 1938.

"The complete Argyll Robertson pupil is one in which the pupil is miotic to the extreme point and there is no reaction to darkness, flash-light or daylight, while the reaction in accommodation is preserved. In our opinion [the authors'], other reflexes are not clinically essential.

"The incomplete Argyll Robertson pupil is one in which there is some dilatation of the pupil. This pupil does not react to flash-light but will widen slowly in the darkness and constrict slowly in a room well illuminated by daylight.

"The light reflex may be restored in a partial way in the Argyll Robertson pupil by instillation of a dilute solution of benzedrine sulfate, ranging from 0.125 to 0.5 per cent, or by repeated subcutaneous injection or oral ingestion of the drug. Under such circumstances the pupil dilates, and while ordinarily only slightly mobile to flash-light, it widens in darkness and constricts in daylight, the movements of dilatation and constriction being slow and deliberate but certain.

"The light reflex, so far as the pupil is concerned, must be considered as made up of two important parts: (1) dilatation of the pupil to darkness or to partial illumination and (2) constriction to light, these movements being brought about by the reciprocal activities of the sympathetic and the parasympathetic nervous system, or, to speak chemically, by a balance between adrenergic and cholinergic substances. Furthermore, part of the light reflex is widening and narrowing of the palpebral fissure, widening taking place in darkness or in lessened illumination and constriction under the influence of light or increased illumination."

R. IRVINE.

Physiologic Optics

CLINICAL RESEARCHES ON THE DIOPTRIC POWER OF THE HUMAN LENS. R. CAMPOS, Ann. di ottal. e clin. ocul. 66:413 (June) 1938.

After a technical consideration of various means of estimating the refractive power of the lens, Campos concludes that the only method at one's disposal is that of studying the change in refraction after removing the lens. On the basis of 15 cases in which the formula of Bierke, as modified by Gullstrand, was employed, Campos found the dioptric power of the lens to vary between 19 and 26 diopters, with an average of 22 diopters.

S. R. GIFFORD.

Physiology

AUTONOMIC INNERVATION OF THE FACE: II. AN EXPERIMENTAL STUDY. F. H. LEWY, ROBERT A. GROFF and F. C. GRANT, Arch. Neurol. & Psychiat. 39:1238 (June) 1938.

In a previous paper the author demonstrated that after section of the oculomotor nerve electrical stimulation of the first division of the fifth nerve is followed by widening of the ipsilateral palpebral fissure

and lacrimation. The muscular contraction was slow and was sustained for a period after electrical stimulation was stopped.

The present work elaborates on this phenomena, showing that the pseudomotor phenomena of the eyelid, the lip, the whiskers and the tongue may be elicited by stimulation of: (1) autonomic efferent fibers in the sensory root or sensory divisions of the fifth nerve originating in the mesencephalic nucleus of the nerve and (2) the preganglionic and postganglionic fibers of the cervical portion of the sympathetic trunk.

They conclude that in the sensory root of the fifth nerve as well as of the third, seventh and twelfth nerves, respectively, efferent fibers pass from the mesencephalic nucleus to the face.

This article should be interesting to ophthalmologists as a suggestion toward the explanation of such bizarre oculomotor phenomena as jaw-winking and the pseudo-Graefe phenomena.

R. Irvine.

Behavior of the Ocular Tension of Normal Persons Under Atmospheric Decompression. A. Bucalossi, Ann. di ottal. e clin. ocul. 66: 292 (April) 1938.

The author examined 12 normal persons from 18 to 24 years of age in a pressure chamber. Pressure was reduced to 490 mm. of mercury at a rate of 13.5 mm. per minute and maintained at the lowest point for fifteen minutes. A slight increase of tension (from 2 to 4 mm. Schiötz) was seen in most of the subjects at the time of minimum pressure. This was parallel with a similar rise of blood pressure and returned to normal when the atmospheric pressure became normal.

S. R. Gifford.

Studies on Dark Adaptation with Automatic Recording of the Threshold Values. W. Kyrieleis, Arch. f. Ophth. 138: 564 (May) 1938.

In 1936 Kyrieleis demonstrated before the German Ophthalmological Society at Heidelberg an attachment to the Engelking-Hartung adaptometer which records on a kymograph the width of the Aubert diaphragm of the adaptometer. With this adaptometer the light thresholds are determined by opening the Aubert diaphragm until the patient sees light. A record of the widths of the diaphragm at these threshold points is, therefore, a record of the respective light sensitivities. With this method it is possible to make ten or more threshold determinations per minute. Dark adaptation curves obtained in this way show a few heretofore unknown details. In the rise of the light sensitivity of the retina during dark adaptation a fairly regular alternation of periods of rapid and slow rise can be observed. The adaptation curve thus appears to consist of numerous short arcs or to describe small steps. The author has studied in detail the effect of exposure to varying degrees of light immediately before dark adaptation and the effect of variations in the size of the pupil. The position of the nick in the adaptation curve is characteristically dependent on the degree of light adaptation prevailing immediately before dark adaptation.

P. C. Kronfeld.

Refraction and Accommodation

STUDIES ON THE HEREDITARY ORIGIN OF MYOPIA. L. PAUL, Arch. f. Ophth. **139:** 378 (Sept.) 1938.

The relationship between the refraction of parents and the refraction of their children is studied statistically in material comprising about 4,000 Germans. The results are shown in the accompanying table.

The clinician distinguishes between low degrees of myopia without any tendency toward chorioretinal complications and high degrees of myopia (myopia gravis) characterized by a definite tendency toward such complication. Paul sets the borderline between the two degrees at a myopia of 10 diopters and finds that the two degrees are transmitted separately and independently of one another. In other words, myopia gravis occurs much more frequently in families in which one parent is affected with myopia gravis than in families in which one parent is affected with a low grade of myopia. On the other hand, myopia of low degree is relatively rare in families in which one parent is affected with myopia gravis.

Statistical Study of Myopia

	A No. ot Families Examined	B No. of Children in These Families	C No. of Children Examined	No. of Myopic Children, Expressed in % of B	No. of Myopic Children, Expressed in % of C
No parent myopic..............	423	1,173	723	7.85	12.72
One parent myopic.............	166	479	297	23.00	37.10
Both parents myopic...........	39	98	72	53.10	72.20

The frequency of myopia of low degree is greater in female than in male infants. During the period of growth this relationship changes, so that (in families with myopia in the ancestry) myopia is more frequent among men than among women. Here the author admits the possibility of the development of myopia through the influence of extrinsic factors. These factors, however, are supposed to be especially effective in persons with myopia in their ancestry.

P. C. KRONFELD.

Retina and Optic Nerve

THE CLINICAL SIGNIFICANCE OF THE RETINAL CHANGES IN THE HYPERTENSIVE TOXEMIAS OF PREGNANCY. G. G. GIBSON, Am. J. Ophth. **21:** 22 (Jan.) 1938.

Gibson gives the following summary of his study of the retinal changes in 39 cases in which a diagnosis of toxemia of pregnancy or of varying degrees of preeclampsia was made:

"The first visible physical sign of early preeclampsia is attenuation of the nasal retinal arterioles. This is usually concurrent with slight elevation of the diastolic blood pressure. The great majority of these cases do not become sufficiently severe to progress further than this stage; they will usually respond to medical treatment as suggested by

Arnold. If they do, the patient will be left without a demonstrable vascular lesion. In a small percentage of the cases, however, the disease is much more fulminating and treatment must be heroic. The earliest and most reliable signs of the severe course of these conditions is the appearance of the retinal arterioles. The severe changes in the retinal arterioles usually precede the clinical criterion of severity in these conditions. The appearance of hemorrhages and exudates associated with hypertensive vascular change in the retinal arterioles is a reliable sign for the therapeutic termination of pregnancy. Proper interpretation of these changes will materially reduce the vascular damage done to the mother, and reduce the incidence of fetal and maternal mortality."

W. S. REESE.

CONTRIBUTION TO THE STUDY OF THE GENESIS OF DETACHMENT OF THE RETINA. E. REDSLOB, Ann. d'ocul. **175:** *637* (Sept.) *1938.*

The vogue for studies on detachment of the retina seems to have abated somewhat. In the last ten years more than 600 articles on this subject have appeared. Have they cleared up all the questions connected with this subject? It seems to the author that though a satisfactory therapeutic technic may have been arrived at, the question of origin is still much to the fore.

The important role attributed to the closing of the tear is known to all ophthalmologists, but in a consideration of whether the tear is the principal etiologic factor in the development of the detachment, different opinions are encountered. Unquestionably there are cases of detachment in which despite the most minute examination with the ophthalmoscope and microscope no sign of a tear is to be found. Such a case of relatively fresh detachment, in which histologic examination eliminated all possibility of perforation of the retina, was reported by the author a few years ago. One may conclude that the mechanism in the development of detachment of the retina is still imperfectly known, and it seems useful to bring together all possible facts in order to arrive at a better understanding of the problem.

The author gives in detail the results of histologic examination of an enucleated globe with two illustrations. S. H. MCKEE.

OXYCEPHALY AND ALTERATIONS OF THE OPTIC NERVE. G. P. SOURDILLE and M. DAVID, Bull. Soc. d'opht. de Paris **50:** 206 (May) 1938.

A boy of 7 had had poor vision for one and one-half years, and for the past six months his sight had been rapidly diminishing. When aged 3 months the child had a severe enteritis with abnormally late closure of the fontanels. His head was of the typical tower skull variety. Normal intelligence but an emotional instability were noted. The visual acuity of the right eye was 2/10 and that of the left eye was 1/12, with correction by a horizontal convex cylinder to 3.5/10 and 1/10, respectively. There was concentric contraction of the visual fields, more marked on the left, to about 40 degrees. The right disk was completely pale, and the left showed pallor only in the temporal region. Roentgenograms of the skull showed the two characteristics of oxycephaly, namely, digital

markings of the convexity, here most formidable, and enlargement of the middle fossa. It is the authors' belief that in this case the atrophy was due to intracranial hypertension. Decompression resulted in vision of 4/10 for the right eye, while the left remained at 1/10 after six weeks. The question of operation in these cases is open for discussion.

L. L. Mayer.

Calcium-Potassium Values of the Blood in Retinal Detachment. A. Bucalossi, Ann. di ottal. e clin. ocul. **66:** 371 (May) 1938.

Previous studies by the author had shown a diminution in the alkaline reserve of patients with retinal detachment. Since potassium is known to favor imbibition of the proteins while calcium inhibits it, possible effects of one or the other ion on the condition of the vitreous are considered. In 10 normal persons the author found an average potassium value of 20.34 mg. per hundred cubic centimeters of blood and a calcium value of 16.44 mg., giving a potassium-calcium quotient of 1.96. The maximum quotient was 2.34 and the minimum 1.60. In 24 patients with idiopathic retinal detachment the average potassium value was 19.11 mg. per hundred cubic centimeters and the average calcium value, 11.14 mg., giving a quotient of 1.71. In 25 per cent of patients the quotient was lower than the lowest of the normal series, while in 75 per cent it was lower than the average of this series. There was no correlation between the potassium-calcium quotient and the alkaline reserve. The increase in calcium might conceivably be a factor in changing the physiochemical constitution of the vitreous in the sense of decreased imbibition of its proteins.

S. R. Gifford.

Bilateral Amaurosis Due to Anatomic and Functional Changes in the Retinal Circulation. C. Gandolfi, Rassegna ital. d'ottal. **7:** 287 (May-June) 1938.

The author reports 2 cases of closure of both central retinal arteries in which careful observation of the condition of the retinal vessels, including determinations with the dynamometer, revealed a different basis for the condition in each case. One case was that of a man of 73 who had suddenly lost vision in each eye two days previously. When he was first seen the vessels were normal in appearance, but vision improved only slightly in spite of repeated injections of acetylcholine. The general blood pressure dropped markedly after the first injection, and the retinal blood pressure became normal. The author considers that the condition was due to spasm of the retinal arteries. In the second case loss of vision in one eye was followed within a day by the occurrence of the same condition in the second eye. The usual fundus picture of closure of the retinal arteries was present in each eye with other signs of arteriosclerosis. Because of these other changes and because the retinal blood pressure remained unvaried at a low level, the condition in this case was interpreted as organic closure of both central arteries, due in all probability to endarteritis and thrombosis. Retrobulbar injections of acetylcholine were of no effect.

S. R. Gifford.

Trachoma

PATHOLOGIC ANATOMY OF THE TRACHOMA FOLLICLE. G. BORSELLO, Rassegna ital. d'ottal. **7:** 361 (May-June) 1938.

The literature on the pathologic anatomy of the trachoma follicle is reviewed, and the author reports his studies of material stained by various methods, including the silver stains and the stains of Giemsa and of Pappenheim. The article is 45 pages in length and does not lend itself to abstracting.

Photomicrographs and a bibliography are included.

S. R. GIFFORD.

ATTEMPTED CULTURE OF THE TRACHOMA AND PARATRACHOMA VIRUS. I. JOHN and F. A. HAMBURGER, Arch. f. Ophth. **138:** 760 (July) 1938.

"Almost exclusively" material (pieces of conjunctiva) from patients with paratrachoma (apparently chiefly cases of inclusion blennorrhea of the newborn) were used for culture experiments, the results of which were disappointing on the whole. Purification of the material, that is, removal of all bacteria, was first done by filtration which entailed dilution of the virus, so that inoculation of baboons with the filtrate had no effect. These filtrates must have contained the virus, because by centrifuging the filtrate at high speed and incubating its sediment together with human embryonic tissue for a few days a material was obtained which microscopically contained bodies similar to the elementary bodies and produced a paratrachoma-like conjunctivitis in the baboon. In various culture mediums which were inoculated with paratrachoma material no Prowazek-Halberstädter inclusions or initial bodies developed, but structures which resembled elementary bodies more or less closely were seen occasionally. Unfortunately, there is no truly specific staining method for elementary bodies. Aside from the intrinsic difficulties of this kind of work, the authors were handicapped by scantiness of clinical material and of experimental animals (baboons).

P. C. KRONFELD.

Tumors

ANNUAL REPORT OF RADIUM INSTITUTE FOR 1937, editorial, Brit. M. J. **2:** 712 (Oct. 1) 1938.

Twenty-eight cases of tumors of the limbus were reported, most of the growths being squamous cell epitheliomas. These were treated by unscreened radium plaques placed in contact with the growth, and no instance of cataract was recorded. In all but 2 cases, in which enucleation was subsequently performed, the results were completely satisfactory.

It is hoped that the patients will be kept under careful observation, so that any possible subsequent effects of irradiation on the retina will be recorded.

ARNOLD KNAPP.

LEUKOSARCOMA OF THE CHOROID: REPORT OF A CASE. G. MATHIS, Rassegna ital. d'ottal. 7: 408 (May-June) 1938.

A patient of 71 had been losing vision in one eye for four years, with recent attacks of pain and hypertension. A gray mass protruded into the anterior chamber through the root of the iris. At enucleation, extension of the neoplasm through the sclera was seen. No pigment was found in the tumor, which was of the mixed cell alveolar type. The intraocular portion was relatively small compared to the orbital extension and was of the flat type.

S. R. GIFFORD.

Uvea

TUBERCULOSIS, SYPHILIS AND CHRONIC ANTERIOR UVEITIS. F. FARINA, Rassegna ital. d'ottal. 7: 301 (May-June) 1938.

Aside from the typical forms of uveitis, which may be rather definitely diagnosed as due to syphilis, tuberculosis or focal infection, the author has interested himself especially in a group of cases in which the uveitis is of the type usually ascribed to various other causes, such as gout, rheumatism, diabetes and intestinal infections. In such cases the uveitis responds poorly to treatment and is prone to recurrence. Farina reports 40 such cases seen during nine years, the patients having been followed from three to five years. In addition to complete physical examination, including serologic tests, tuberculin tests and an analysis of the blood chemistry, he has in many cases examined the aqueous microscopically and considers his observations of considerable value. The presence of lymphocytes and a large number of crystals is considered evidence in favor of a tuberculous origin. In cases in which the uveitis is due to syphilis, polymorphonuclear leukocytes and pigment granules are found in larger numbers, with fewer crystals. Two cases are reported in which the diagnosis of syphilis was made on this basis and the Wassermann reaction, previously negative, became positive after antisyphilitic treatment. The patients had no recurrences during further observation. In another case a diagnosis of tuberculosis was made chiefly because of changes in the aqueous, and the administration of tuberculin resulted in apparent cure. Although only 4 cases are reported in detail, the author states that similar events occurred in many of the other cases, the serologic tests or the cutaneous tests becoming positive during treatment.

S. R. GIFFORD.

Society Transactions

EDITED BY W. L. BENEDICT

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

ALEXANDER G. FEWELL, M.D., *Chairman*

Dec. 15, 1938

WARREN S. REESE, M.D., *Clerk*

IRIDESCENT CRYSTALS IN THE CRYSTALLINE LENS. DR. ALEXANDER G. FEWELL and DR. STIRLING S. MCNAIR.

The literature has been reviewed with special reference to cases in which the crystalline lens contained iridescent crystals. The presence of ordinary crystals in the lens is not uncommon, but the occurrence of iridescent crystals is indeed rare. In most of the reported cases of iridescent crystals in the lens the crystals contained some calcium salt, protein or one of the amino acids. In our search through the literature we are able to find only 1 case, that reported by Beresinskaja, in which chemical analysis proved cholesterol crystals to be present. In a majority of the cases reported the iridescent crystals were found bilaterally. Although these crystals are supposedly due to degenerative changes in a cataract, the majority of the case reports state that the remaining portion of the lens is clear. It is surprising how few lenses containing iridescent crystals have been extracted and examined chemically.

The case reported here is that of a Negress aged 69 who had bilateral anterior polar cataract in addition to bilateral senile cataract. She had had diabetes for the past four years and also suffered from hypertension. The vision in the right eye was 3/60 and in the left eye 2/60. Examination of the right eye with the slit lamp revealed large masses of brownish golden glistening crystals, somewhat tightly packed together in places, more dense and numerous in the center and on the temporal side. These crystals were mostly in the stroma of the lens, and the remainder of the lens was relatively clear. The fundus was seen with difficulty on account of these crystals and the opacities of the lens. The disk was somewhat dusky, the arteries were reduced in caliber and some retinal changes were seen in the macular area but no hemorrhages. The left eye showed a mature cataract but no crystals. The fundus could not be seen. We expect to extract the lens of the right eye and subject its crystals to microchemical examination. We hope to report the results of this examination at a later date.

DISCUSSION

DR. ALFRED COWAN: The case which I reported here three or four months ago was different from this one. In my case the crystals were brightly colored—red, green, yellow and orange—and were more or less

densely arranged in a zone in and around the adult nucleus. Actually, presenile, zonular cataract which had undergone degeneration was present. I would consider the condition in the case reported an atypical complicated cataract.

Dr. William Zentmayer: Some years ago Dr. Verhoeff reported a case of coralliform cataract in which a thorough analysis of the crystals of which the opacity was formed showed them to be protein. Soon after this report I removed a lens with the same type of cataract and sent it to Dr. Verhoeff. The analysis showed the same type of crystals as was found in his own case.

Essential Shrinking of the Conjunctiva (Ocular Pemphigus): Pemphigus of the Mucous Membranes. Dr. Joseph V. Klauder and Dr. Van M. Ellis.

R. H., a white youth aged 21, was unable to state exactly when the present condition started, but he had had ocular trouble at the age of 4 years. At this age he was said to have had a growth on the right eye. When he was examined at the age of 21 the right cornea was clear. There was adhesion of the conjunctiva of the lower lid to the bulbar conjunctiva, obliterating the cul-de-sac. The upper cul-de-sac was narrowed; there was no adhesion of the conjunctiva of the upper lid to the bulbar conjunctiva. The left cornea also was clear, and there was no involvement of the conjunctiva. The cul-de-sacs were, however, narrowed. The vision was unimpaired. There was no involvement of the mucous membranes of the nose, mouth, throat, urethra or anus. There had never been any cutaneous involvement.

Old Interstitial Keratitis. Dr. Joseph V. Klauder and Dr. Van M. Ellis.

A white man, aged 58, came to the clinic because of poor vision. He had always had poor vision and had worn glasses for many years. He was an only child. He remembered having sore eyes when a child but could not recall any details. He said that he had never had a genital lesion. After a "blood" test ten years previously he had received injections in the arm at intervals for three months.

The pupils were unequal and irregular in outline. The right pupil was fixed to light, and the left reacted slightly. Gross inspection of the corneas showed faint opacities. The vision with correction was 6/15 in the right eye and 6/30 in the left eye. Slit lamp examination of the right eye revealed the central portion of the cornea to be gray with a moderate loss of smoothness. The posterior surface was thickened and fairly opaque and contained a number of old blood vessels, deeply situated. Examination of the left eye gave essentially the same results. The patellar reflexes were absent, and the Romberg sign was positive. The patient presented no stigmas of congenital syphilis, except very suggestive facies, and he had no subjective symptoms of tabes. The Wassermann reaction of the blood was 4 plus; the Meinicke reaction was 2 plus. Examination of the spinal fluid gave negative results in all phases.

This case is reported to call attention to the importance of being conscious of the cornea in examining patients of any age suspected or

known to have syphilis. This applies more to the clinician and the syphilologist than to the ophthalmologist, who is more cognizant of the significance of old corneal opacity as a clue in the diagnosis of old interstitial keratitis than is any other specialist.

Corneal examination, especially slit lamp microscopy, may be an important procedure in examination of a patient, especially an adult with a positive Wassermann reaction. These examinations may be the only means of determining that the patient has congenital syphilis.

Effective Treatment of Lupus Erythematosus and Exudative Retinitis with a Gold Compound. Dr. Joseph V. Klauder and Dr. Van M. Ellis.

E. D., a white man aged 36, was first seen in April 1938, at which time he exhibited on each side of the face and on the forehead bright red areas ranging in size from that of a dime to that of a half dollar. Some of the areas were infiltrated, and all were devoid of scales. The scalp and mucous membranes were not involved. Ophthalmoscopic examination revealed various-sized, irregular, fluffy, cloudlike patches scattered throughout the fundus of the right eye, particularly around the disk. There were three small fresh hemorrhages around the macula. The appearance of the left eye was the same as that of the right eye, but the involvement was not so extensive. Vision was 3/60 in each eye. Three months prior to the time he was first seen the patient had chills, fever, general malaise, weakness and tiredness, which necessitated rest in bed. About that time an eruption developed on the face. A month later he noticed blurring of vision. He was incapacitated from January 1938 until about April because of progressive weakness and loss of 23 pounds (10.4 Kg.) in weight. There was no history of tuberculosis; the patient had always been underweight but of good general health. The Mantoux reaction to a 1:100 dilution of tuberculin was negative. Roentgenograms of the lungs were negative, as were the results of examination of the sputum for tubercle bacilli. Urinalysis gave negative results, and the Wassermann reaction of the blood was negative. The blood sugar content was 71.7 mg. per hundred cubic centimeters; the urea, 13.6 mg., and the uric acid, 3.1 mg. A complete blood count on March 26 showed: red blood cells, 4,640,000; white blood cells, 3,400; polymorphonuclears, 46 per cent; eosinophils, 1 per cent; lymphocytes, 45 per cent, and monocytes, 8 per cent. On April 1 the white blood cells numbered 6,200 and on April 4, 8,300. Histologic examination showed subacute diffuse dermatitis and atrophy of the epidermis with local hyperplasia.

Beginning April 1938, the patient was treated with gold and sodium thiosulfate intravenously (the maximum dose, 50 mg.) and bismuth salicylate (2.5 cc.) intramuscularly. These drugs were given in courses at weekly intervals. Apparently the treatment was effective. The cutaneous lesions disappeared, and the patient gained 15 pounds (6.8 Kg.) in weight and is no longer tired. The appearance of the fundi improved. At present the retinitis is quiescent. The optic disk has a pale white tint and is well outlined. Some of the larger vessels have been replaced by scar tissue; other vessels are sclerosed and have a corkscrew appearance. The vision in each eye is 6/60. Collateral circulation was observed over each disk.

DISCUSSION

Dr. Walter I. Lillie: In regard to the case of lupus erythematosus, examination of the fundi revealed a diffuse perivasculitis and periphlebitis associated with pallor of the disks and the formation of new blood vessels around and involving each disk. Although a retinitis specific for lupus erythematosus has never been described, the retinitis observed before the therapy with gold and sodium thiosulfate was instituted could have been of toxic origin.

The present change in the fundi is similar to that which occurs after the intravenous administration of quinine in a person with an idiosyncrasy to quinine.

In my experience, gold and sodium thiosulfate has produced no untoward ocular effects and has been efficacious in the treatment of posterior uveal disease.

Dr. H. Maxwell Langdon: I should like to ask Dr. Klauder whether he thinks there have been any progressive changes in the case of pemphigus since he first saw the patient. Frankly, the condition does not look like pemphigus to me. I have never seen pemphigus limited to the lower cul-de-sac and have never seen it last so long without the other eye becoming involved. On the basis of a history of ocular trouble at 4 years of age, it looks to me as though the condition might be scarring due to the inflammation seventeen years ago rather than pemphigus.

Concerning the patient with interstitial keratitis, has Dr. Klauder thought of the possibility of acquired syphilis? I admit that the likelihood is slight, but such cases do occur.

Dr. Joseph V. Klauder: We are glad to know Dr. Langdon's opinion regarding the case of suspected pemphigus. We are inclined to regard the condition as progressive. It appears to be more pronounced now than at the time we first saw the patient. In formulating our opinion of ocular pemphigus in this patient, we were not entirely influenced by the presence of symblepharon. We placed considerable significance on the dry, lusterless, wrinkled appearance of the bulbar conjunctiva external to the cornea and above the symblepharon. The appearance was that of shrinking, and it is a conspicuous symptom eventually in all patients with ocular pemphigus. However, we will keep the diagnosis open pending further observation.

Regarding the cases of interstitial keratitis, I have never observed interstitial keratitis in a patient with acquired syphilis. Igersheimer has stated that when syphilis is acquired in infancy or childhood interstitial keratitis is likely to appear in adult life. Other authors have expressed the belief that when chancre involves the conjunctiva interstitial keratitis is likely to appear subsequently. In many of the reported cases of interstitial keratitis occurring in association with acquired syphilis that I have reviewed, I did not regard the evidence sufficient to prove that syphilis was acquired. For example, a history of a genital lesion is no proof that that lesion was a chancre. In still other reports there was no slit lamp evidence to support the diagnosis of interstitial keratitis.

Production of Corneal Ulcers in the Rabbit. Dr. Robb McDonald and Dr. Horace Pettit.

This paper is published in full in this issue of the Archives, page 817.

VARIED SURGICAL INDICATIONS OF PTOSIS. DR. EDMUND B. SPAETH.

The various operative indications in any case of ptosis are the factors which modify the type of operation to be used. No one procedure can or will answer more than one set of indications and conditions. Therefore, it is necessary to utilize various procedures: those involving the occipito-frontalis muscle and at other times the superior rectus muscle or the superior oblique muscle in the direction of the ptosis. (Lantern slides were shown demonstrating the appearance before and after correction in representative cases.)

Jan. 19, 1939

FINAL OCULAR RESULT IN A CASE OF ANTERIOR CEREBELLAR-BULBAR-PONTILE POLIOMYELOENCEPHALITIS, TWENTY-SEVEN YEARS AFTER THE ACUTE ATTACK. DR. EDWARD A. SHUMWAY.

A woman, aged 33 was admitted to the University Hospital in September 1911, to the service of Dr. Charles K. Mills, at that time professor of neurology in the University of Pennsylvania. Neurologic examination showed complete paralysis of the left facial nerve and right hemiplegia. The tongue deviated to the right and the jaw to the left. There was paralysis also of the fifth and sixth cranial nerves and of the associated movements of the eyes, both to the right and to the left and up and down.

Ocular examination showed slight reduction of vision but entirely normal eyegrounds.

Dr. Mills' final diagnosis as the result of observations extending over years was "poliomyelitis, destructive, cerebellar-bulbar and pontile."

For a time the presence of a tumor was considered, and operation was at one time discussed, but the idea was dismissed. The patient had been under my care in my private practice for twenty-seven years.

As a result of the paralysis of the sixth nerve, which, with the involvement of the seventh nerve, remained permanent, and the weakness of the associated movements, both lateral and vertical, right hyperphoria and esotropia had developed. The hyperphoria had increased from 2 to 6 degrees as a result of the turning upward and inward of the left eye. The esophoria had decreased from 8 to 6 degrees. After final stabilization of the muscular condition, the patient was made comfortable with 3 degree prisms, base up in the left eye and base down in the right eye, for distance and prisms of 2 degrees in the same positions in each eye for reading. The esophoria did not need correction, and the patient was free from diplopia, both for distance and for near vision.

The final report of the neurologic condition, by Dr. J. W. McConnell, made a few days before this meeting, showed absence of sensory paralyses; recovery from the right hemiplegia, except for some hyperactivity of the deep reflexes on the right side, and permanent paralysis of the left facial nerve, which had been partly relieved by suturing the eyelids at the outer canthus. Finally, as a remaining sign

of bulbar involvement there was paresis of the left half of the vocal cords, which was confirmed by Dr. Ralph Butler and was due to a motor disturbance of the left recurrent laryngeal nerve.

DISCUSSION

Dr. Walter I. Lillie: Dr. Shumway is to be envied in having had the privilege of observing such a case over a period of twenty-seven years. It does not fall to one's lot to be able to do this often. The case is obviously one of poliomyelitis, because of the history and the fact that the residual defects are entirely motor. Localization is obviously correct, because it included DeFoville's syndrome, which is paralysis of the sixth and seventh nerves with contralateral hemiplegia. The patient had also a homolateral Horner's syndrome, which fits into this picture. It has always been interesting that these patients do not have involvement of the eighth nerve, in spite of the fact that auditory nuclei are situated in the vicinity of the lesion. I believe that if the hearing could be examined like the visual fields, a homonymous hearing defect would be found in a case of this sort.

Dr. Edward A. Shumway: I think that I should mention Dr. Butler's report in regard to the ears.

He said that at present the patient's hearing on the left side is four tenths of normal and that this deafness is due to involvement of the middle ear, probably the result of a former suppurative inflammation, as there is a scar on the ear drum. Deafness may have been due to involvement of the eighth nerve, however, as Dr. Lillie has suggested.

My reasons for presenting this case are to report the ability to make the patient comfortable from an ophthalmologic point of view by the use of vertical prisms and to stimulate interest among ophthalmologists in neuro-ophthalmology.

Adie's Syndrome: Report of a Case. Dr. Harold G. Scheie.

It is important to recognize Adie's syndrome and to differentiate it from tabes dorsalis with Argyll Robertson pupils. Since in the case observed by me the pupil contracted well with physostigmine sulfate and was sensitized to choline compounds, it was felt that a partial denervation of the sphincter might have occurred.

DISUSSION

Dr. Francis Heed Adler: Kinnier Wilson stated that no one should make a diagnosis of Argyll Robertson pupils unless he had read Argyll Robertson's original paper. It is certain that the diagnosis of Argyll Robertson pupils has been incorrect many times because all of the characteristics of this phenomenon were not strictly adhered to. Adie's syndrome is a case in point. One cannot be certain at the present time where the site of the lesion is in cases of Adie's syndrome, but with the help of animal experimentation it is possible that Dr. Scheie will be able to locate it more accurately.

DR. WARREN S. REESE: Are patients with this syndrome entirely symptomless?

DR. I. S. TASSMAN: Was the patient's accommodation tested after the use of choline?

DR. HAROLD G. SCHEIE: The patient has no symptoms, because only the ciliary muscle of one side is affected. If both sides were affected, the patient would certainly complain of blurring with changing the distance of his gaze.

This patient was tested before and after the use of choline, but I found little or no change from its use.

VARIOUS TYPES OF OCULAR MOVEMENTS: THEIR CLINICAL SIGNIFICANCE. DR. A. BIELSCHOWSKY, Hanover, N. H.

There are four main groups of ocular movements: the voluntary (commanded) movements; the attraction movements, which are brought about by sensorial stimuli attracting attention; the fusion movements, and the reflex movements of vestibular origin. The second and third groups are called psycho-optic reflexes because they take place almost automatically, without awareness of intention on the part of the subject; however, they are not true reflexes, as they are elicited by sensorial stimuli only if the latter attract attention.

Through error, doubts have been expressed as to the strict validity of the law according to which under normal conditions every innervation flows equally to the two eyes. The fact that a single eye can be moved separately does not prove that the unilateral movement is produced by a unilateral innervation. If two impulses are sent to the eyes simultaneously, one driving them in the same direction and the other in the opposite direction, an unequal or even unilateral movement must result. An ocular movement is never brought about by one single muscle. All the ocular muscles always cooperate in performing any ocular movement, one half acting as agonists and the other as antagonists. The same cortical center which controls the contraction of the agonists brings about the active relaxation of the antagonists, so that a movement may take place even if the agonists are unable to obey this motor impulse because of total paralysis.

Fusion movements are important for the visual act, particularly in persons with heterophoria, which can be kept latent only as long as the fusion mechanism maintains the innervation compensating the anomalous position of the eyes relative to each other.

The only true reflex movements of the eyes originate in the vestibular apparatus. Every change of the position of the head or body causing a current of the endolymph in the semicircular canals causes a stimulation which is conveyed to the motor nuclei by the posterior longitudinal bundle. The reaction of the ocular muscles aims at keeping the position of the eyes in space unaltered, notwithstanding the change of the position of the head or body.

A characteristic vestibular reaction is presented by the parallel rotary movement of the eyes around their anteroposterior axes, elicited by the tilting of the head toward the opposite side. This reaction is at the basis of ocular torticollis, which in cases of palsy of the trochlear

nerve and other disturbances of the oblique muscles helps the patient to see single. The head tilting test (Bielschowsky and Hofmann) is described as a method of diagnosing disturbances of the vertical motor muscles of the eyes.

The reaction of the eyes to vestibular stimuli is most important in cases of palsy of the supranuclear type. Failure of the muscles to respond to voluntary impulses while they react to vestibular stimuli produced by my head rotation test or by Bárány's test is proof that the nuclei and the peripheral nerve fibers are intact. (The characteristics of attraction or following movements were discussed with regard to those cases of palsy of the supranuclear type in which only the following movements are obtainable. The importance of Bell's phenomenon was illustrated by reports of cases of paralysis of the associated upward movement in which this phenomenon was the only proof of the supranuclear site of the lesion.) According to the particulars which can be ascertained by the various methods of investigation, it may be possible to make an approximate localization of the lesion causing the associated paralysis in an individual case.

Die erblichen Hornhautdystrophien. Dystrophiae corneae hereditariae. By Prof. Dr. Max Bücklers, Tübingen. Price, 6.80 marks. Pp. 143, with 86 illustrations, 12 family trees and 3 tabulations. Stuttgart: Ferdinand Enke, 1938.

In this exhaustive publication Bücklers presents a study of 129 personal cases of hereditary corneal dystrophy, observed in and around Württemberg, a country in Southern Germany comparable in size and population with New Jersey. He has uncovered 12 family trees, some going back to the middle of the seventeenth century and comprising 300 persons. This rich material has been thoroughly sifted, and as a result of his painstaking studies the author was able to settle definitely some pertinent questions.

The conclusions arrived at by him are here presented.

All hereditary corneal dystrophies can be reduced to three independent pathologic conditions: (1) granular (nodular) corneal dystrophy (dystrophia corneae granularis [Groenouw I, Fleischer and others]); (2) macular corneal dystrophy (dystrophia corneae macularis [Groenouw II, Fuchs, Fehr, Fleischer and others]), and (3) lattice-like corneal dystrophy (dystrophia corneae reticulata [Haab, Dimmer and others]).

1. Granular corneal dystrophy is a dominant hereditary disease, beginning in the first decade of life in the form of minute changes visible only by the slit lamp. Later these changes become larger. They occupy a disklike area corresponding to a pupil of medium width. The intermediate tissue and a marginal zone always remain clear. Visual acuity does not suffer much, and what impairment there is occurs later in life; because of this, patients need not be sterilized.

2. Macular corneal dystrophy is a recessive hereditary disease. It, also, begins in the first ten years of life as a more or less diffuse and more rapidly progressing opacity of the whole cornea. The changes are macroscopically visible between the twentieth and the thirtieth year of life; they are of a more diffuse character than in the granular variety and impair the vision much more. Persons affected with macular dystrophy are to be sterilized.

3. Lattice-like corneal dystrophy is a dominant hereditary disease. It, too, begins in the first decade of life. In contrast to the opacities of the two other varieties, optical disturbances (lacunar formations or hyaline ridges) are responsible for its "lattice fibers." Later a disciform opacity may form in the center. Abrasions of the cornea are frequent. The surface becomes irregular early in youth, and vision suffers accordingly. The lattice-like dystrophy is much rarer than either the granular or the macular type. Persons suffering from the lattice-like type should be sterilized.

Hereditary corneal dystrophies all begin in early childhood and are always binocular. Both sexes are equally affected.

The opacities develop simultaneously in the two eyes and are frequently symmetric. They progress and resist all medication.

There are no transitional forms between the three types of corneal hereditary dystrophy, neither in the same person nor in the same family. There is no connection between these corneal dystrophies and other hereditary conditions.

The numerous pictures which adorn the book, some in color and most of them enlarged and somewhat overdone in order to show more impressively the essential clinical features, are especially valuable when they demonstrate the earliest stages of the disease, which are easily overlooked; the importance of the slit lamp in the study of these early phases is stressed.

A short discussion of all the reported cases precedes the description of the author's own cases. The various inefficient therapeutic measures are recorded. The microscopic anatomic picture of the lesions will be dealt with in a later publication.

A complete bibliography concludes the book, the acquisition of which is highly commended to anybody who is interested in one of the most engaging pathologic conditions of the cornea.

ERNST WALDSTEIN.

Anais da Faculdade de Medicina de Pôrto Alegre. Volume 1, July-September, 1938. Rio Grande do Sul, Brazil: Universidade de Pôrto Alegre.

The first number of a new quarterly journal has recently appeared which is published by the members of the Faculty of Medicine of Pôrto Alegre. In this number of 300 pages there are two articles by ophthalmologists.

The first article is by Dr. H. Arruga on the treatment of detachment of the retina. He considers the fundamental principle of all operative methods for this condition to be Gonin's great discovery that the tear should be closed. The percentage of recoveries with this method, has been increased from 25 to 75 per cent in a few years. In cases in which no tear can be determined the detachment results from a choroidal exudate, and the treatment consists in removal of the inflammatory cause.

There are two important methods for localization. With the first, a blunt diathermy tip is used to make a small area of coagulation on the sclera which can be recognized with the ophthalmoscope. With the second method, that of Weve, a mark is made on the sclera at the place where the ophthalmoscope illuminates the pathologic retinal area. In general, the blunt diathermy tip is now preferred, and the sharp electrode is used only to facilitate the escape of the subretinal fluid.

With the new methods of operating, satisfactory results have been obtained in cases of extensive tears in the periphery of the retina, in cases of detachment at the ora serrata and in cases of holes in the macular region. The author has operated on 810 patients, with 396 clinical cures.

The second article is by Prof. Corrêa Meyer and deals with biologic problems of hereditary conditions in ophthalmology. He gives a general survey on the present knowledge of heredity based on the investigations of authors from Mendel to Morgan. The hereditary diseases in man are described, particularly as to their forms of heredity in connection with ophthalmology.

The conception of abiotrophy is particularly studied. According to the author, in such cases lethal semilethal factors are transmitted directly according to the mendelian law, whereby the cells of greatest development are first attacked and undergo a premature degeneration.

Finally, the author discusses from social standpoints the question of the prevention of hereditary diseases.

ARNOLD KNAPP.

Manual de enfermedades de los ojos. By Prof. Carlos E. Finlay, with the collaboration of Thomas Yanes, M.D. Pp. 664, with 275 black and white illustrations and 19 color plates. Habana, Cuba: "Cultural," S. A., 1939.

This manual, the author claims, has been written mostly for the benefit of medical students, general practitioners and those commencing the study of ophthalmology. A series of lectures delivered to medical students by Professor Finlay has been used for the publication of this book, with the addition of a few chapters contributed by Dr. Yanes. The manual contains twenty chapters, written in clear style. The authors have not tried to be exhaustive but have presented only the fundamentals of ophthalmology. The classic surgical procedures have been dealt with in detail. Other less known surgical methods have been briefly mentioned.

The book contains 275 illustrations in black and white and 19 color plates. Many of the illustrations, especially the photographs, are original, taken of personally observed patients of Dr. Finlay and of some of his Cuban colleagues. The color illustrations of fundi have been taken from the Atlas of Oller and are well reproduced.

Dr. Finlay emphasizes that in order to institute therapeutic methods properly, a solid foundation of anatomy, physiology and histopathology should be laid. For this reason therapy has been dealt with more extensively than is generally found in manuals of this type.

The relation between ocular and general diseases, as well as those of the nervous system, are well and simply outlined. The chapters on refraction, ocular hygiene, workmen's injuries and ocular therapeutics are clearly and concisely dealt with by Dr. Yanes. Most of the chapter on anomalies of the muscles has been taken from the Duane translation of Fuchs's "Text-Book of Ophthalmology," for which acknowledgment is made.

This book is dedicated to the memory of Dr. Finlay's parents, Carlos J. Finlay and Adela Shine de Finlay, and to Prof. Herman Knapp, for whom Dr. Finlay has great admiration, their friendship dating back to the days when he was an assistant at the New York Ophthalmic and Aural Institute.

RAMÓN CASTROVIEJO.

Twelfth Annual Report of the Giza Memorial Ophthalmic Laboratory, Cairo, 1937. Price, 35 piasters. Pp. 150. Cairo: Schindler's Press, 1938.

In addition to the clinical work of the laboratory, Major F. H. Stewart is carrying on experimental work on the causation of trachoma and Dr. F. Maxwell Lyons is conducting a careful investigation into factors bearing on the causation and treatment of spring catarrh. During the Fifteenth International Ophthalmologic Congress, which was held in Cairo, Dec. 8-14, 1937, the laboratory was visited by many members. During 1937 two postgraduate courses were given on medical and surgical ophthalmology, and 565 pathologic specimens were examined. Among these there were 91 eyes lost as the result of purulent ophthalmia. There were 68 specimens showing malignant tumors and 76 showing lesions due to trachoma. The presence of eosinophils is not regarded as important for the diagnosis of spring catarrh in Egypt, while the thin glistening tenacious membrane which forms on the tarsal conjunctiva on exposure to the air is a most valuable clinical sign.

A number of interesting pathologic reports and reports of unusual clinical cases are given. The results of treating trachoma with sulfanilamide and primary glaucoma with extract of adrenal cortex were unsatisfactory. The appendix contains studies on the pathologic picture of experimental trachoma by F. H. Stewart and on the biomicroscopic picture of spring catarrh by F. Maxwell Lyons.

The director, Dr. Rowland P. Wilson, is to be congratulated on the continued excellence of the work done in this laboratory.

ARNOLD KNAPP.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Dr. P. Bailliart, 66 Boulevard Saint-Michel, Paris, 6e, France.
Secretary-General: Prof. M. Van Duyse, Université de Gand, Gand, Prov. Ostflandern, Belgium.
All correspondence should be addressed to the Secretariat, 66 Boulevard Saint-Michel, Paris, 6e, France.

International Ophthalmologic Congress

President: Prof. Nordenson, Serafimerlasarettet, Stockholm, Sweden.
Secretary: Dr. Ehlers, Jerbanenegade 41, Copenhagen, Denmark.

International Organization Against Trachoma

President: Dr. A. F. MacCallan, 33 Welbeck St., London, W., England.

FOREIGN

All-India Ophthalmological Society

President: Dr. B. K. Narayan Rao, Minto Ophthalmic Hospital, Bangalore.
Secretary: Dr. G. Zachariah, Flitcham, Marshall's Rd., Madras.

British Medical Association, Section on Ophthalmology

President: Dr. Stewart Duke-Elder, 59 Harley St., London, W. 1.
Secretary: Dr. Thomasina Belt, 13 Mitchell Ave., Jesmond, Newcastle-on-Tyne.

Chinese Ophthalmology Society

President: Dr. C. H. Chou, 363 Avenue Haig, Shanghai.
Secretary: Dr. F. S. Tsang, 221 Foochow Rd., Shanghai.

Chinese Ophthalmological Society of Peiping

President: Dr. H. T. Pi, Peiping Union Medical College, Peiping.
Secretary: Dr. C. K. Lin, 180 Hsi-Lo-yen, Chienmeng, Peiping.
Place: Peiping Union Medical College, Peiping. Time: Last Friday of each month.

German Ophthalmological Society

President: Prof. W. Lohlein, Berlin.
Secretary: Prof. E. Engelking, Heidelberg.

Hungarian Ophthalmological Society

President: Prof. H. G. Ditroi, Szeged.
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, Maria ucca 39, Budapest.
All correspondence should be addressed to the Assistant Secretary.

Midland Ophthalmological Society

President: Dr. W. Niccol, 4 College Green, Gloucester, England.
Secretary: T. Harrison Butler, 81 Edmund St., Birmingham, England.
Place: Birmingham and Midland Eye Hospital.

Ophthalmological Society of Egypt

President: Prof. Dr. Mohammed Mahfouz Bey, Government Hospital, Alexandria.
Secretary: Dr. Mohammed Khalil, 4 Baehler St., Cairo.
All correspondence should be addressed to the Secretary, Dr. Mohammed Khalil.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. T. Harrison Butler, 81 Edmund St., Birmingham, England.
Secretary: Mr. L. H. Savin, 7 Queen St., London, W. 1, England.

OPHTHALMOLOGY SOCIETY OF BOMBAY

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4, India.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14, India.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. Percival J. Hay, 350 Glossop Rd., Sheffield 10, England.
Time: July 6-8, 1939.

PALESTINE OPHTHALMOLOGICAL SOCIETY

President: Dr. Arieh Feigenbaum, Abyssinian St. 15, Jerusalem.
Secretary: Dr. E. Sinai, Tel Aviv.

POLISH OPHTHALMOLOGICAL SOCIETY

President: Dr. W. Kapuściński, 2 Waly Batorego, Poznań.
Secretary: Dr. J. Sobański, Lindley'a 4, Warsaw.
Place: Lindley'a 4, Warsaw.

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Dr. Malcolm Hepburn, 111 Harley St., London, W. 1, England.
Secretary: Dr. C. Dee Shapland, 15 Devonshire Pl., London, W. 1, England.

SOCIÉTÉ FRANÇAISE D'OPHTALMOLOGIE

Secretary: Dr. René Onfray, 6 Avenue de la Motte Picquet, Paris, 7e.

SOCIETY OF SWEDISH OPHTHALMOLOGISTS

President: Prof. K. G. Ploman, Stockholm.
Secretary: Dr. K. O. Granström, Södermalmstorg 4 III tr., Stockholm, Sö.

TEL AVIV OPHTHALMOLOGICAL SOCIETY

President: Dr. D. Arieh-Friedman, 96 Allenby St., Tel Aviv, Palestine.
Secretary: Dr. Sadger Max, 9 Bialik St., Tel Aviv, Palestine.

TSINAN OPHTHALMOLOGICAL SOCIETY

Chairman: Dr. Eugene Chan, Cheeloo University School of Medicine, Tsinan, Shantung, China.
Place: Cheeloo University School of Medicine. Time: Last Thursday of alternate months.

NATIONAL

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY

Chairman: Dr. S. Judd Beach, 704 Congress St., Portland, Maine.
Secretary: Dr. Derrick T. Vail Jr., 441 Vine St., Cincinnati.
Place: St. Louis. Time: May 15-19, 1939.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, SECTION ON OPHTHALMOLOGY

President: Dr. Harry S. Gradle, 58 E. Washington St., Chicago.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Oct. 8-13, 1939.

American Ophthalmological Society

President: Dr. Frederick Tooke, 1482 Mountain St., Montreal, Canada.
Secretary-Treasurer: Dr. Eugene M. Blake, 303 Whitney Ave., New Haven, Conn.
Place: Hot Springs, Va.

Canadian Medical Association, Section on Ophthalmology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal.
Secretary-Treasurer: Dr. J. A. MacMillan, 1410 Stanley St., Montreal.
Place: Montreal. Time: June 19-23, 1939.

National Society for the Prevention of Blindness

President: Mr. William Fellowes Morgan, 50 W. 50th St., New York.
Secretary: Miss Regina E. Schneider, 50 W. 50th St., New York.

SECTIONAL

Central Wisconsin Society of Ophthalmology and Otolaryngology

President: Dr. R. O. Ebert, 104 Main St., Oshkosh.
Secretary: Dr. G. L. McCormick, 626 S. Central Ave., Marshfield.
Place: Oshkosh. Time: May 1939.

New England Ophthalmological Society

President: Dr. Edwin B. Goodall, 101 Bay State Rd., Boston.
Secretary-Treasurer: Dr. Trygve Gundersen, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time. 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. F. C. Cordes, 384 Post St., San Francisco.
Secretary-Treasurer: Dr. C. Allen Dickey, 450 Sutter St., San Francisco.
Place: San Francisco. Time: June 19-22, 1939.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. Edward Clark, 1305–14th Ave., Seattle.
Secretary-Treasurer: Dr. Purman Dorman, 1115 Terry Ave., Seattle.
Place: Seattle or Tacoma, Wash. Time: Third Tuesday of each month, except June, July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. L. J. Friend, 425 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. Thorsten E. Blomberg, 501–7th St., Rockford, Ill.
Place: Rockford, Ill., or Janesville or Beloit, Wis. Time: Third Tuesday of each month from October to April, inclusive.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. Don M. Howell, Alma, Mich.
Secretary-Treasurer: Dr. Louis D. Gomon, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw or Bay City, Mich. Time: Second Tuesday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. R. A. Kelly, 304 N. Main St., Mitchell, S. D.
Secretary-Treasurer: Dr. J. C. Decker, 515 Frances Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Grady E. Clay, Medical Arts Bldg., Atlanta, Ga.
Secretary: Dr. John R. Hume, 921 Canal St., New Orleans.

Southwestern Michigan Triological Society

President: Dr. Carl Wencke, Battle Creek.
Secretary-Treasurer: Dr. A. K. Zinn, Battle Creek.
Time: Third Thursday of alternate months.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. M. Harris, Johnstown.
Secretary-Treasurer: Dr. C. Wearne Beals, 41 N. Brady St., DuBois.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: Dr. Edward Jackson, 1008-A Republic Bldg., Denver.
Secretary: Dr. John C. Long, 324 Metropolitan Bldg., Denver.
Place: University Club, Denver. Time: 6:30 p. m., third Saturday of each month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. William M. Good, 63 Center St., Waterbury.
Secretary-Treasurer: Dr. S. J. Silverberg, 201 Park St., New Haven.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Grady E. Clay, 384 Peachtree St., N. E., Atlanta.
Secretary-Treasurer: Dr. J. Mason Baird, 511 Medical Arts Bldg., Atlanta.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. C. W. Rutherford, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Marlow W. Manion, 23 E. Ohio St., Indianapolis.
Place: Indianapolis. Time: First Wednesday in April.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. H. Lamb, American Bank Bldg., Davenport.
Secretary-Treasurer: Dr. B. M. Merkel, 604 Locust St., Des Moines.
Place: Davenport.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Francis E. Le Jeune, 632 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Place: Gulfport, Miss. Time: May 8, 1939.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. B. Fralick, 201 S. Main St., Ann Arbor.
Secretary: Dr. O. McGillicuddy, 124 W. Allegan St., Lansing.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Frank N. Knapp, 318 W. Superior St., Duluth.
Secretary-Treasurer: Dr. George E. McGeary, 920 Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. Roy Grigg, Bozeman.
Secretary: Dr. A. W. Morse, 507 Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Norman W. Burritt, 30 Beechwood Rd., Summit.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: Atlantic City. Time: June 1939.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. Algernon B. Reese, 73 E. 71st St., New York.
Secretary: Dr. Chester C. Cott, 333 Linwood Ave., Buffalo.

North Carolina Eye, Ear, Nose and Throat Society

President: Dr. F. C. Smith, 106 W. 7th St., Charlotte.
Secretary-Treasurer: Dr. M. R. Gibson, Professional Bldg., Raleigh.
Place: Statesville. Time: Sept. 21, 1939.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. N. A. Youngs, 322 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516–6th St., Valley City.
Place: Fargo. Time: May 1939.

Oregon Academy of Ophthalmology and Oto-Laryngology

President: Dr. L. O. Clement, 406 State St., Salem.
Secretary-Treasurer: Dr. Paul Bailey, 833 S. W. 11th Ave., Portland.
Place: Good Samaritan Hospital, Portland. Time: Third Tuesday of each month.

Rhode Island Ophthalmological and Otological Society

Acting President: Dr. N. Darrell Harvey, 112 Waterman St., Providence.
Secretary-Treasurer: Dr. Linley C. Happ, 124 Waterman St., Providence.
Place: Rhode Island Medical Society Library, Providence. Time: 8:30 p. m., second Thursday in October, December, February and April.

South Carolina Society of Ophthalmology and Otolaryngology

President: Dr. S. B. Fishburne, 1430 Marion St., Columbia.
Secretary: Dr. J. W. Jervey Jr., 101 Church St., Greenville.

Tennessee Academy of Ophthalmology and Otolaryngology

President: Dr. J. B. Stanford, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. W. D. Stinson, 124 Physicians and Surgeons Bldg., Memphis.

Texas Ophthalmological and Oto-Laryngological Society

President: Dr. A. N. Champion, 705 E. Houston St., San Antonio.
Secretary: Dr. Dan Brannin, 1719 Pacific Ave., Dallas.
Place: Houston. Time: December 1939.

Utah Ophthalmological Society

President: Dr. V. P. White, 143½ S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. E. B. Fairbanks, Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

Virginia Society of Oto-Laryngology and Ophthalmology

President: Dr. Charles T. St. Clair, 418 Bland St., Bluefield, W. Va.
Secretary-Treasurer: Dr. M. H. Williams, 30½ Franklin Rd., S. W., Roanoke.

West Virginia State Medical Association, Eye, Ear, Nose and Throat Section

President: Dr. George Traugh, 309 Cleveland Ave., Fairmont.
Secretary: Dr. Welch England, 621½ Market St., Parkersburg.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

President: Dr. Andrew Rados, 31 Lincoln Park, Newark.
Secretary: Dr. William F. McKim, 317 Roseville Ave., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month, October to May.

Akron Academy of Ophthalmology and Otolaryngology

President: Dr. L. E. Brown, Second National Bldg., Akron, Ohio.
Secretary-Treasurer: Dr. C. R. Anderson, 106 S. Main St., Akron, Ohio.
Time: First Monday in January, March, May and November.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. Arthur G. Fort, 478 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. Lester A. Brown, 478 Peachtree St., N. E., Atlanta, Ga.
Place: Grady Hospital. Time: 6:00 p. m., second Wednesday of each month from October to May.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Frank B. Walsh, Wilmer Institute, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. Fred M. Reese, 6 E. Eager St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Birmingham Eye, Ear, Nose and Throat Club

President: Each member, in alphabetical order.
Secretary: Dr. N. E. Miles, 408 Medical Arts Bldg., Birmingham, Ala.
Place: Tutwiler Hotel. Time: 6:30 p. m., second Tuesday of each month, September to May, inclusive.

Brooklyn Ophthalmological Society

President: Dr. E. Clifford Place, 59 Livingston St., Brooklyn.
Secretary-Treasurer: Dr. Frank Mallon, 1135 Park Pl., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. Ivan J. Koenig, 40 North St., Buffalo.
Secretary-Treasurer: Dr. Meyer H. Riwchun, 367 Linwood Ave., Buffalo.
Time: Second Thursday of each month.

Chattanooga Society of Ophthalmology and Otolaryngology

President: Each member, in alphabetical order.
Secretary: Dr. A. H. Benz, 706 Medical Arts Bldg., Chattanooga, Tenn.
Place: Mountain City Club. Time: Second Thursday of each month from September to May.

Chicago Ophthalmological Society

President: Dr. Georgiana Dvorak-Theobald, 715 Lake St., Oak Park, Ill.
Secretary-Treasurer: Dr. Earle B. Fowler, 55 E. Washington St., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cincinnati Ophthalmic Club

Chairman: Each member, in rotation.
Secretary-Treasurer: Dr. E. R. Thomas, 819 Carew Tower, Cincinnati.
Place: Holmes Memorial Library, Cincinnati General Hospital. Time: 8:15 p. m., third Monday of each month except June, July and August.

Cleveland Ophthalmological Club

Chairman: Dr. Paul Moore, Republic Bldg., Cleveland.
Secretary: Dr. G. Leslie Miller, 14805 Detroit Ave., Cleveland.
Time: Second Tuesday in October, December, February and April.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Clerk: Dr. W. S. Reese, 1901 Walnut St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Hugh G. Beatty, 150 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. W. A. Stoutenborough, 21 E. State St., Columbus, Ohio.
Place: Deshler Wallick Hotel. Time: 6 p. m., first Monday of each month.

Corpus Christi Eye, Ear, Nose and Throat Society

Chairman: Dr. Edgar G. Mathis, 416 Chaparral St., Corpus Christi, Texas.
Secretary: Dr. E. King Gill, 416 Chaparral St., Corpus Christi, Texas.
Time: Second Thursday of each month from October to May.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. Maxwell Thomas, Medical Arts Bldg., Dallas, Texas.
Secretary: Dr. J. Dudley Singleton, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. E. G. Linn, 604 Locust St., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month from September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Arthur S. Hale, 1609 Eaton Tower, Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. L. A. Hulsebosch, 191 Glen St., Glen Falls.
Secretary-Treasurer: Dr. Joseph L. Holohan, 330 State St., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. A. Gough, 602 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Charles R. Lees, 806 Medical Arts Bldg., Fort Worth, Texas.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Dewey R. Heetderks, 405 Medical Arts Bldg., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Robert G. Laird, 116 E. Fulton St., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. Louis Daily, 1215 Walker Ave., Houston, Texas.
Secretary: Dr. Herbert H. Harris, 1004 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. J. K. Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to June.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. E. N. Robertson, Concordia, Kan.
Secretary: Dr. John S. Knight, 1103 Grand Ave., Kansas City, Mo.
Time: Third Thursday of each month from October to June. The November, January and March meetings are devoted to clinical work.

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

Chairman: Dr. Ben K. Parks, 619 Professional Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Paul Nilsson, 211 Cherry Ave., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Secretary-Treasurer: Dr. John P. Lordan, 2007 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:00 p. m., fourth Monday of each month from September to May, inclusive.

LOUISVILLE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Gaylord C. Hall, Brown Bldg., Louisville, Ky.
Secretary-Treasurer: Dr. Charles K. Beck, Starks Bldg., Louisville, Ky.
Place: Brown Hotel. Time: 6:30 p. m., second Thursday of each month from September to May, inclusive.

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Earle Breeding, 1801 I St., N. W., Washington.
Secretary: Dr. Elmer Shepherd, 1606–20th St., N. W., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order.
Secretary: Dr. Sam H. Sonders, Medical Arts Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. John E. Mulsow, 231 W. Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. John B. Hitz, 411 E. Mason St., Milwaukee.
Place: University Club. Time: 6:30 p. m., second Tuesday of each month.

MINNEAPOLIS OPHTHALMOLOGICAL SOCIETY

Chairman: Each member, in alphabetical order.
Secretary: Dr. M. C. Pfunder, 645 Medical Arts Bldg., Minneapolis.
Place: Hennepin County Medical Society rooms. Time: 6:30 p. m., fourth Monday of each month, October to May, inclusive.

MONTGOMERY COUNTY MEDICAL SOCIETY

Chairman: Dr. H. V. Dutrow, 1040 Fidelity Medical Bldg., Dayton, Ohio.
Secretary-Treasurer: Dr. Maitland D. Place, 981 Reibold Bldg., Dayton, Ohio.
Place: Van Cleve Hotel. Time: 6:30 p. m., first Tuesday of each month from October to June, inclusive.

Montreal Ophthalmological Society

President: Dr. L. de G. Joubert, 690 Dunlop St., Montreal, Canada.
Secretary: Dr. K. B. Johnston, 1509 Sherbrooke St., W., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Guy Maness, 119–7th Ave., Nashville, Tenn.
Secretary-Treasurer: Dr. Andrew Hollabaugh, Doctors Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to May.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. E. G. Walls, 619 Maison Blanche Bldg., New Orleans.
Secretary-Treasurer: Dr. W. B. Clark, 1012 American Bank Bldg., New Orleans.
Place: Louisiana State University Medical Bldg. Time: 8 p. m., second Tuesday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. James W. White, 15 Park Ave., New York.
Secretary: Dr. Rudolf Aebli, 30 E. 40th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

New York Society for Clinical Ophthalmology

President: Dr. Percy Fridenberg, 38 W. 59th St., New York.
Secretary: Dr. David Alperin, 889 Park Pl., Brooklyn.
Place: Squibb Hall, 745–5th Ave. Time: 8 p. m., first Monday of each month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Philip Romonek, 107 S. 17th St., Omaha.
Secretary-Treasurer: Dr. W. Howard Morrison, 1500 Medical Arts Bldg., Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Ophthalmological Society of the University of Pittsburgh

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Time: Second Monday in November, January, March and May.

Passaic-Bergen Ophthalmological Club

President: Dr. R. N. Berke, 430 Union St., Hackensack, N. J.
Secretary-Treasurer: Dr. T. A. Sanfacon, 340 Park Ave., Paterson, N. J.
Place: Paterson Eye and Ear Infirmary. Time: 9 p. m., last Friday of every month, except June, July and August.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Walter I. Lillie, 255 S. 17th St., Philadelphia.
Secretary: Dr. Edmund B. Spaeth, 1930 Chestnut St., Philadelphia.
Time: First Thursday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward Stieren, Union Trust Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, 351–5th Ave., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. Rudolph Thomason, Professional Bldg., Richmond, Va.
Secretary: Dr. Richard W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

ROCHESTER EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edwin S. Ingersoll, 26 S. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. Charles T. Sullivan, 277 Alexander St., Rochester, N. Y.
Place: Rochester Academy of Medicine, 1441 East Ave. Time: 8 p. m., second Wednesday of each month from September to May.

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. B. Y. Alvis, Carleton Bldg., St. Louis.
Secretary: Dr. Carl C. Beisbarth, 3720 Washington Blvd., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

SAN ANTONIO OPHTHALMOLO-OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Oscar H. Judkins, 414 Navarro St., San Antonio, Texas.
Secretary-Treasurer: Dr. Wilfred E. Muldoon, 414 Navarro St., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Russell Fletcher, 490 Post St., San Francisco.
Secretary: Dr. Avery Morley Hicks, 490 Post St., San Francisco.
Place: Society's Bldg., 2180 Washington St., San Francisco. Time: Fourth Tuesday of every month except May, June, July and December.

SHREVEPORT EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. R. R. Kirkpatrick, 6th and Walnut Sts., Texarkana, Ark.
Secretary-Treasurer: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Place: Shreveport Charity Hospital. Time: 7:30 p. m., first Monday of every month except July, August and September.

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. P. B. Greene, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. O. M. Rott, 421 Riverside Ave., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

SYRACUSE EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. James F. Cahill, 428 S. Salina St., Syracuse, N. Y.
Secretary-Treasurer: Dr. I. Herbert Katz, 713 E. Genesee St., Syracuse, N. Y.
Place: University Club. Time: First Tuesday of each month except June, July and August.

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan, 170 St. George St., Toronto, Canada.
Secretary: Dr. W. R. F. Luke, 170 St. George St., Toronto, Canada.
Place: Academy of Medicine, 13 Queens Park. Time: First Monday of each month, November to April.

WASHINGTON, D. C., OPHTHALMOLOGICAL SOCIETY

President: Dr. G. Victor Simpson, 1710 Rhode Island Ave., N. W., Washington, D. C.
Secretary-Treasurer: Dr. Frank D. Costenbader, 1726 I St., Washington, D. C.
Place: Episcopal Eye, Ear and Throat Hospital. Time: 8 p. m., first Monday in November, January, March and May.

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